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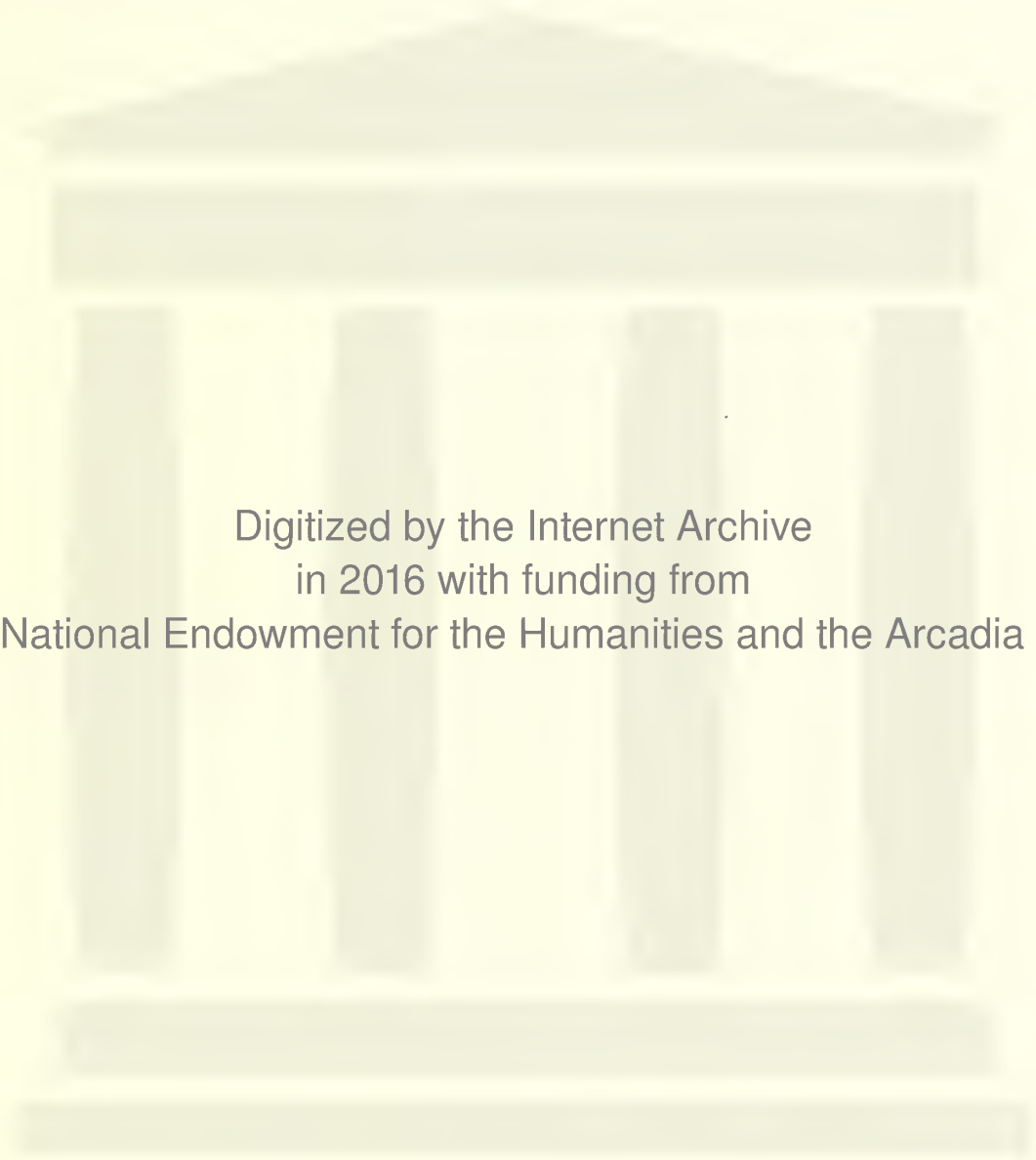

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Published Monthly by the S.D. State Medical Assn.

Volume XXXVII January 1984 Number 1

SOUTH DAKOTA JOURNAL OF Medicine

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Clinicopathologic Conference Thirty Year Old Caucasian Male With Acute Onset of Abdominal Pain

Atrioventricular Block — A Review

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References:
1. Stone PH, Turi ZG, Muller JE. Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104 672-681, September 1982.
2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302 1269-1273, June 5, 1980.

BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug Interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis. Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request.

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Quotes from an unsolicited letter received by Pfizer from an angina patient. While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

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"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).

for the varied faces of angina

* Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

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J-4044 January 1984

Due to a printer's error, pages 13 thru 16 were inadvertently omitted from our December, 1983 issue.

These pages, along with the four pages completing the scientific article entitled, ''Epidermal and Intrathecal Narcotics for Pain Relief,'' are reprinted in this issue beginning on page 5.

We apologize for any confusion or inconvenience this may have caused our readers.

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SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 1 January 1984 (ISSN 0038-3317)

Official Publication of the South Dakota State Medical Association

the South Dakota Chapter of The American Academy of Family Physicians

the South Dakota Chapter of the American College of Surgeons

the South Dakota Psychiatric Association of the American Psychiatric Association

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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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Clinicopathologic Conference
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HISTORY OF THE BLACK HILLS SUMMER SEMINAR

During the 1960s, the Black Hills Medical Society (9th District of the South Dakota State Medical Association) developed and sponsored a Black Hills Seminar, providing continuing medical education for area physicians.

Dr. Paul Aspaas of Dell Rapids was President of the South Dakota Academy of Family Physicians in 1967-68 and then served as Secretary-Treasurer from 1968-71, while Dr. Donald L. Scheller of Arlington was President. During this time of service, Dr. Aspaas noted a decline of the Black Hills Seminar, which he had attended and enjoyed. Realizing the need for quality continuing education for Academy members, he sought support to resurrect this meeting under the direction of SDAFP. At that time, the American College of OB/GYN had money available for CME efforts, and Dr. Aspaas enlisted the aid of Dr. Buck Williams of Sioux Falls, an officer in the South Dakota Chapter of ACOG in securing funds and co-sponsoring the first Black Hills Summer Seminar in 1970. This ACOG co-sponsorship lasted for several years. Dr. Lloyd Sweeney of Sioux Falls, then an officer in the South Dakota Academy, and later President 1971-73, supported this effort and helped with the development of the scientific program, recently completing its 14th year.

Recognition of this history was made during the 1983 Black Hills Summer Seminar.

RESIDENCY PROGRAM REACCREDITED

The Sioux Falls Family Practice Residency Program had a re-accreditation visit from the Residency Review Committee for Family Practice in April of 1983.

The RRC-FP is composed of representatives of the AAFP, ABFP, and the AMA Council on Medical Education and represents the Accreditation Council for Graduate Medical Education (ACGME) in reviewing family practice residency programs.

Full re-accreditation for five years, with no restrictions, was received early in August of 1983.

Congratulations!

PATIENT EDUCATION TIPS

High Blood Pressure Control Is Worth Its Salt

Salt is a chemical, made up of 40 percent sodium. Although the body must have sodium, most Americans eat much more of it than they need. This can be harmful, because sodium seems to contribute to higher blood pressures in some people. Many of those who have high blood pressure can help control it by reducing the amount of sodium they eat. And a low sodium diet can help some people keep health blood pressure from becoming high.

What can patients do to cut down on sodium? Use more fresh or frozen (unsauced) produce and meats, avoid processed foods and foods that taste salty, and don't add salt in cooking, baking, or at the table. Read the labels on all packaged foods to check for salt or sodium. And remember, a taste for salt is a habit; the less one uses, the less one wants.

Advise your patients what role watching sodium might play in controlling blood pressure and staying healthy.

FAMILY PRACTICE CLUB AT USDSM

The annual SDAFP sponsored Fall Family Practice Club meeting at USDSM was held on Friday evening, November 4, at the Newman Club Center in Vermillion. Nearly 170 people attended this event, including medical students and spouses from all classes, 4 Family Practice Residents and spouses from the Sioux Falls program, and 10 SDAFP active members and spouses.

SDAFP is appreciative of Marion Labs cosponsorship of this and other Family Practice Club events planned for the year. Medical students will be guests of SDAFP at the 9th annual Black Hills Winter Ski Seminar in Spearfish, February 2-4, 1984. Residency Day, tentatively scheduled for Friday, May 4, 1984 will feature activities for third year students including input from family practice residents.

December

President's Page

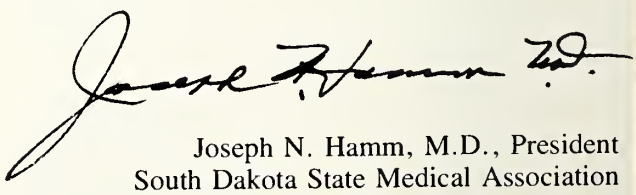
Gaudeamus Igitur

The song begins, "Gaudeamus igitur, iuvenes dum sumus"; and ends "Post iucundum iuventutem, post molestam senectutem, nos habebit humus —". The words were written by a medieval poet, now forgotten, and became a drinking song in the universities of Europe. The song has not been forgotten. Many who read this page learned the song in glee club or choir during student days. Freely translated the Latin reads; "Therefore let us rejoice, while we are young; — after a delightful youth, after an irksome old age, the grave will contain us —."

Youth has always been cherished by those who possess it and its passing has been lamented down the corridors of time. Yet American children are being robbed of youth. Child abuse has reached disaster proportions in America — even in our own state. The U. S. Department of Health and Human Services estimates that 1.8 million children are missing annually. They are being tortured and killed. Some who survive the physical abuse are left with minds too worn to cope. What youth is left is wasted in confinement.

Physicians should lead the way in stamping out the epidemic of child abuse. The Council on Scientific Affairs of the AMA has adopted a three year program and has recommended it to the state associations. Our own auxilliary has joined its national body in implementing a prevention program. Our Governor has directed the power of his office to combat child abuse. Physicians have the knowledge, skill and compassion to support the effort. We must make the commitment.

A MERRY CHRISTMAS AND A BOUNTIFUL, PEACEFUL 1984 TO ALL!



Joseph N. Hamm, M.D., President
South Dakota State Medical Association

Epidural and Intrathecal Narcotics for Pain Relief

Edward F. Anderson, M.D.*

"Thousands of Americans with advanced cancer are living the last months of their lives in severe pain. Available pain medications are more than adequate to relieve their suffering, but many physicians are reluctant to prescribe them in sufficient dosages or for long enough intervals."

The above statement was not made 40 years ago, but in January, 1983 at a national medical conference in Washington, D.C. by the president of the AMA, William Y. Rial. He went on to say, "It is the responsibility of every physician and others who serve patients to understand the dynamics of pain, to understand the pharmacologic activity of analgesics, and, most importantly, to work with and understand the needs of each patient and the family."[1]

Furthermore, the dilemma of today's cancer patients is that while modern chemotherapeutic agents, radiation, and other medical therapy have prolonged their lives, the degree and extent of their pain have also increased.[2] According to John Bonica, not many physicians are trained to view **acute** pain as a diagnostic aid and are thus ill-prepared for the medical management of chronic pain.[1]

As long as man has existed, there has been pain. In the days of primitive man, pain was thought to be due to demons, and the medicine man or shaman, while uttering magical incantations, would fight the invisible demons, thus relieving the patient of his pain. The medicine man was eventually replaced by the priest in the church of the early middle ages who relied on prayer, as well as methods such as alcohol and the administration of the soporific sponge, for pain relief. Subsequently, many drugs were used to induce artificial sleep for pain relief and operations. These included henbane, poppy, mandragora, and hemp and were compounded from many herbs, roots, barks, berries, seeds and blossoms of flowers. Opium had also been inhaled, ingested and applied locally to wounds for its analgesic and hypnotic effects from biblical times until the 19th century. Variability in dosage was one of the major drawbacks of its use.[3]

Serturner, in 1809, isolated morphine as the most active of the more than 20 alkaloids in opium. The invention of the syringe by Pravay and needle by Wood in 1853 allowed administration of narcotics intramuscularly in measured doses. Meperdine was synthesized in 1939, and fentanyl was introduced by Janssen in 1962. When narcotics were administered in large doses for the relief of chronic pain, respiratory depression was almost always present and at times lethal.[4] In addition to the use of oral and parenteral narcotics (such as the Brompton mixture, **ad libitum)** for relief of pain due to chronic malig-

* Staff Anesthesiologist, Member of the Anesthesia Department and Pain Clinic, Sioux Valley Hospital, Sioux Falls, South Dakota, 57105. Address given to the South Dakota Association of Nurse Anesthetists' 33rd Annual Spring Meeting, Sioux Falls, South Dakota, May 1, 1983.

nancy, more recent modalities of therapy have been introduced: chemotherapy, endocrine therapy, radiation therapy, and regional pain control with neurolytic regional blocks or neurosurgical posterior rhizotomy. Controversial therapies for the treatment of chronic cancer pain include: intrathecal cold saline, hypertonic saline, cerebrospinal fluid (C.S.F.) barbotage, acupuncture, T.E.N.S., dorsal column stimulation, percutaneous column stimulation, and subarachnoid ammonia salts, with varying results.[5]

In 1979, the use of intrathecal and epidural morphine was first described in the treatment of patients with chronic pain and produced intense and prolonged analgesia.

In 1973, Pert and Snyder reported on the discovery of opioid receptors in the nervous system, and in 1976 Yakish and Rudy first reported on the intrathecal administration of morphine.[6, 7] This was given to rats, was associated with profound analgesia, and had no adverse tissue reaction in the spinal cord.[7, 8] In 1979, the use of intrathecal and epidural morphine was first described in the treatment of patients with chronic pain and produced intense and prolonged analgesia.[9, 10] More recently, these techniques have been advocated and found extremely useful in treating those patients with acute pain.[11, 12]

Narcotics have always been assumed to operate in the central nervous system (C.N.S.). Narcotic receptors, however, were not able to be identified since narcotics bind to most biologic membranes. Recent techniques using radioactive opiates resulted in separation of binding sites allowing identification of these receptors. It was found that the body produces smaller peptides, enkephalins, and larger peptides, endorphins or endogenous morphine-like substances, which bind to opiate receptors within the C.N.S. and are associated with the production of analgesia.[13]

Opiate receptors are found in very high concentrations in the periaqueductal gray matter of the brain stem, amygdala, corpus striatum and hypothalamus. These receptors are not only involved with pain perception and integration, but also emotional responses to pain. Naloxone, a narcotic antagonist, applied to the periaqueductal gray matter can block the effect of morphine.[14] Opiate receptors are also found in the substantia gelatinosa of the dorsal horn of the spinal cord. It is thought that morphine acts on receptors in Rexed's laminae I, II, and V in the dorsal horn. The small A-delta and C fibers which transmit pain are thought to synapse in laminae I and V whereas nerves carrying other types of sensation, such as touch, synapse with nerves in laminae IV and VI.[15]

When injected into the spinal fluid, narcotics will have the advantage of bypassing the blood brain barrier and then diffusing from the aqueous phase in the cerebrospinal fluid to the lipid phase of the neuraxis.

Narcotics are complex, 3 dimensional compounds which exist as two optical isomers, only the L-form of which is able to produce analgesia. There is a relationship between the structure and analgesic activity. Minor changes in the structure such as the degree of ionization may result in variation of analgesic activity. Narcotics produce analgesia by acting on pre- and post-synoptic membranes of small cell networks both in the spinal cord and higher centers. So called "side effects" such as depression of gastric secretions, cough reflex, and vasomotor tone are due to stimulation of solitary nuclei, which receive visceral sensory fibers from the ninth and tenth cranial nerves, in the brain stem. Nausea and vomiting is due to stimulation of the area postrema with its chemoreceptor trigger zone. Narcotics, like local anesthetics, are ionizable cations, have ionization constants, and pass through lipid and aqueous layers in the C.N.S. before reaching their anionic target They exhibit both specific and nonspecific binding to various tissues. Specific binding occurs at the opiate receptor sites and is reversed by naloxone in a dose dependent manner.[4, 16]

The analgesic effect following epidural morphine, however, correlates poorly with serum morphine levels and remains profound for 18 hours to several days.

Narcotics with strong binding characteristics will outlast the 1-1½ hour duration of naloxone and may reestablish their effect. When injected into the spinal fluid, narcotics will have the advantage of bypassing the blood brain barrier and then diffusing from the aqueous phase in the cerebrospinal fluid to the lipid phase of the neuraxis.[17] Narcotics and local anesthetics have comparable molecular weights and ionization constants that are close to physiologic pH allowing each to pass fully between the aqueous and lipid phases of tissue barriers. Morphine, unlike local anesthetics, has a low degree of lipid solubility indicating that it is slow to pass out of the C.S.F. into the lipid tissues. The oil/water ratio will determine the speed of penetration, onset, duration, spread in the C.S.F. and washout. Receptor binding strength

will also affect duration.[18] The loss of effect in the C.S.F. is due to clearance. The more lipid soluble the drug, the quicker it is cleared.[16]

After the initial reports in animal studies and man, volunteer studies on pain-free humans were carried out by Bromage to determine their effects. In one study injection of methadone and dilaudid into the thoracic and lumbar epidural space caused intense segmental analgesia with minimal respiratory depression or urinary retention.[19] When Bromage compared 10 mg. morphine administered intravenously versus the lumbar epidural route, he found greater analgesia and more side effects in the epidural route. Analgesia was initially confined to the lower extremity and after 3-4 hours spread cephalad to involve the upper extremities and even the face in 50% of the subjects. This lasted 16-22 hours.[20] Samii, with epidural morphine, found pruritus occurring at 3 hours, nausea at 4 hours, and vomiting at 6 hours. As with respiratory depression the above occurred in a dose dependent manner and could also be reversed with naloxone in a dose dependent manner.[21]

There are two stages of respiratory depression with morphine. The early stage is usually seen within one hour and is secondary to vascular uptake from the epidural space. This stage would not likely be seen with intrathecal administration (Figure 1). The late stage, which can be seen with both routes, is

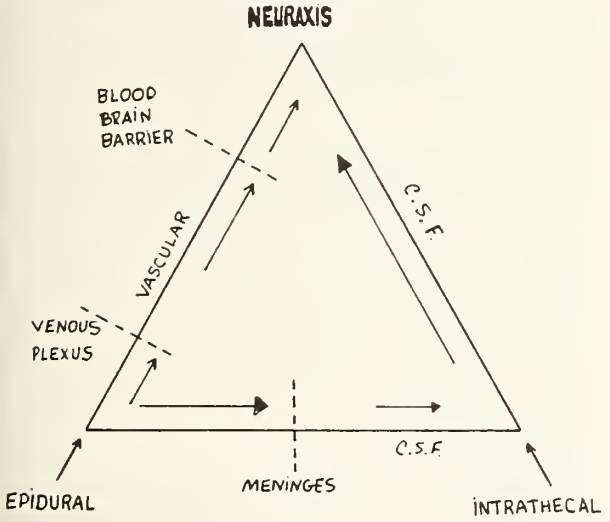


Figure 1
Schematic diagram demonstrating relationship between epidural and intrathecal injections. The epidural injection can result in some absorption into the blood through the internal vertebral venous plexus possibly causing an early stage of respiratory depression. Parenteral narcotics must bypass the blood-brain barrier to be effective. Most of the epidural injection will enter the cerebrospinal fluid through the dural root sleeves and exert its effect on the neuraxis. The intrathecal injection directly enters the cerebrospinal fluid and exerts its effect. Late respiratory depression can occur because of cephalad spread.

related to the lipid/water solubility of the narcotic and to cephalad spread by C.S.F. flow within the subarachnoid and ventricular systems. Respiratory depression is greatest 6-10 hours after injection but may be seen as late as 23 hours. Naloxone will reverse the depression, but repeated doses probably will be necessary.[22, 23, 24] Present evidence suggests that the lipid soluble narcotics methadone, meperidine, and fentanyl diffuse from the C.S.F. to spinal cord lipids rapid enough to probably avoid the cephalad spread seen with the poorly soluble morphine.[18]

Intraspinal (i.e. epidural and intrathecal) narcotics are being used in the treatment of acute (postoperative, obstetric, post-traumatic) and chronic (cancer, other debilitating illness) pain.[11, 16] Bromage, in a recent review, summarized the results of many studies on the use of these techniques. Analgesia is dose dependent and initially segmental. Larger doses cause greater analgesia and duration. When fentanyl (highly lipid soluble) is injected into the epidural space, onset is rapid, distribution is segmental, and duration is short. Morphine (poorly lipid soluble) injection, however, results in slow onset, more general distribution, and prolonged duration (16-24 hours vs. 1-2 hours for fentanyl).[18] Other narcotics have intermediate durations of action (Table I). Serum levels following epidural in-

Table I
The doses and durations of narcotics when administered in 10 cc. balanced salt solution into the epidural space.[16]

Drug	Lipid Sol	Dose	Analgesia
Fentanyl	high	0.1 mg.	3 hrs.
Levodromeran	moderate	4 mg.	6 hrs.
Meperidine	moderate	100 mg.	9 hrs.
Morphine	low	5 mg.	10 hrrs.
		10 mg.	24 hrs.

jections of morphine closely parallel those of intramuscular injections. The analgesic effect following epidural morphine, however, correlates poorly with serum morphine levels and remains profound for 18 hours to several days.[25] Chronic pain is easier to control than acute pain. Smaller doses of narcotics can be used. Intrathecal morphine can control labor pains whereas the epidural route is not as effective. Generally, 1-2 mg. morphine intrathecally can relieve visceral pain during the first stage of labor without any effect on established labor or newborn. However, if repeat injections were necessary during the second stage, an epidural approach with a catheter in place would be more practical.[11]

In order to minimize the occurrence of respiratory depression, dosage should be restricted to the minimal amount necessary for analgesia.[21] Injecting

It is important to note that when narcotics are injected into the intrathecal and epidural compartments, the preservative-free form of the drug must be used.

as close to the dermatone level of pain one wishes to control and placing the patient in a head-up tilt while injecting would also be helpful.[12, 21] Because respiratory depression is inversely related to the length of the vertebral column, it would be less likely in taller patients for comparable doses.[26]

Generally, the intrathecal route would be best when the narcotic could be combined with a spinal anesthetic or only a single injection would be necessary (as diagnostic relief of chronic or cancer pain to determine the suitability of a catheter implantation or post-operative pain relief when injected at the end of a surgical procedure with the patient anesthetized). Prolonged administration of intraspinal narcotics is done through an insertion of an indwelling catheter. The catheter can be safely employed through either extradural or intrathecal approaches. A permanent intrathecal catheter can be extended subcutaneously, lateral from the subarachnoid space, and be connected to a reservoir (e.g., Cordis drug infusion reservoir) (Figure 2). The reservoir is commonly placed subcutaneously subcostally about the level of the anterior axillary line, and its location allows for easy injection by nursing personnel or family members at home.[27] One-half to two mg. doses of morphine are injected Q8-12 hours. Smaller doses, as given with the intrathecal approach, appear to limit respiratory depression and more frequent injections seem to result in less development of tolerance.[26, 28]

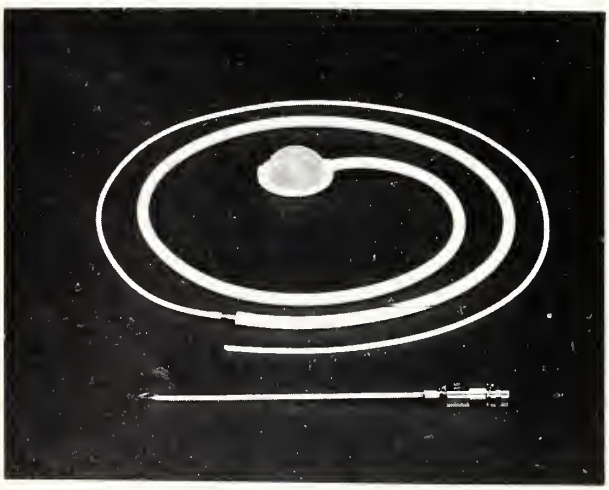


Figure 2
The Cordis drug infusion reservoir used for chronic intrathecal narcotic injections. (Drug infusion reservoir. Number 9EO458 Photograph courtesy of Cordis Corporation, Miami, Florida, 33152).

The epidural route can also be used. For postoperative pain, a standard epidural catheter can be used and left in place for several days, if necessary. Dannemiller recommends the use of the wire-wound Racz catheter for epidural insertion since it is easy to handle and unlikely to kink.[28] In addition, it is designed to be used without a removable stylet, has a "spring like" tip to prevent inadvertent dural puncture, and is designed to be left in the epidural space for extended periods of time.[29] Although a definite advantage in the treatment of long term pain, there was a recent report in some patients of difficulty removing these catheters.[30] Malignancy pain can be controlled with smaller doses of morphine in the epidural space such as 2-4 mg. Postoperative or acute pain may require 6-8 mg. morphine. It is important to note that when narcotics are injected into the intrathecal and epidural compartments, the preservative-free form of the drug must be used. Preservatives, such as phenol, sodium formaldehlyde sulfoxylate, and metacresol, could be neurotoxic. Although tubex syringes are classified as "preservative-free," these should not be used for intraspinal injections.[31] Techniques involving intermittent injections and continuous infusions of narcotics through an implanted reservoir for chronic treatment have been studied.[32, 33] The use of a volumetric infusion pump has been found to be very helpful and can eliminate the inconvenience of reinjecting over a long period of time.[34] It may well be that the acute pain or trauma and surgery will, in the future, be best controlled with a continuous infusion of a lipid soluble narcotic.[14]

The key to safe management of these patients lies with proper monitoring of vital signs, most importantly respiration.

In the hospital, these patients should be monitored in an "intensive care-like" setting post-injection.[18, 27] With many major surgical cases such as thoracotomies, aortobifemoral bypasses, multisystem trauma, etc., this would not be a problem since they would ordinarily be admitted to the Intensive Care Unit (ICU). Other surgical, as well as chronic pain, patients, however, might not otherwise have to be admitted to the I.C.U. Here an intermediate care unit (such as post-coronary) would be helpful and would save I.C.U. space for the more critically ill patients. Once a proper dose regimen is established in these patients and their vital signs have remained stable without any evidence of respiratory depression, they are transferred to their referring ward or home. On the average, this requires 24-72 hours. Nursing personnel, responsible

It is important to note that the use of intraspinal narcotics should not be accompanied by parenteral narcotic administration.

family members, and patients are instructed in proper injection technique as well as potential problems and treatment. When a narcotic is being infused continuously into the epidural space, the patient should remain in the intermediate care setting as long as the infusion is running.[18]

The key to **safe management** of these patients lies with proper monitoring of vital signs, most importantly respiration. Respiratory rate is a poor index of depression and apneic episodes may occur with little warning. Apneic monitors have not been found to be helpful or reliable in these patients.[35] A standing order should be written so that when respiratory depression or other side effects are present, naloxone should be administered immediately intravenously by the nurse (0.1-0.2 mg. dose). It is important to note that the use of intraspinal narcotics should **not** be accompanied by parenteral narcotic administration.[12]

The use of intraspinal naracotics is revolutionary and has many possible future benefits. It can provide pain relief of longer duration with smaller doses in patients having acute and chronic pain previously resistent to other treatment. Intraspinal narcotics are a mode of treatment to be considered along with the other modalities previously mentioned but not necessarily replacing them. It remains for us in anesthesiology and other specialties interested in pain management to determine which patients can be properly controlled by a specific method and when more comprehensive data is available to make specific recommendations.

We still do not have all the answers to many questions on intraspinal narcotics. Hopefully, however, the above information has provided insight into a promising area of pain control and will be useful in determining which patients might benefit from this new mode of pain therapy. In conclusion, Bromage has stated regarding this technique: "Accumulating evidence suggests that intelligent manipulation and exploitation of relevant physicochemical and pharmacological variables will lead to the development of rational clinical regimen that will be both safe as well as more effective and controllable than other analgesic options."[12]

ACKNOWLEDGMENT

The author would like to express his appreciation to Richard G. Belatti, M.D. and William J. Horner, M.D., for their critique of the article and to John Olson, Sioux Valley Hospital, for his photography.

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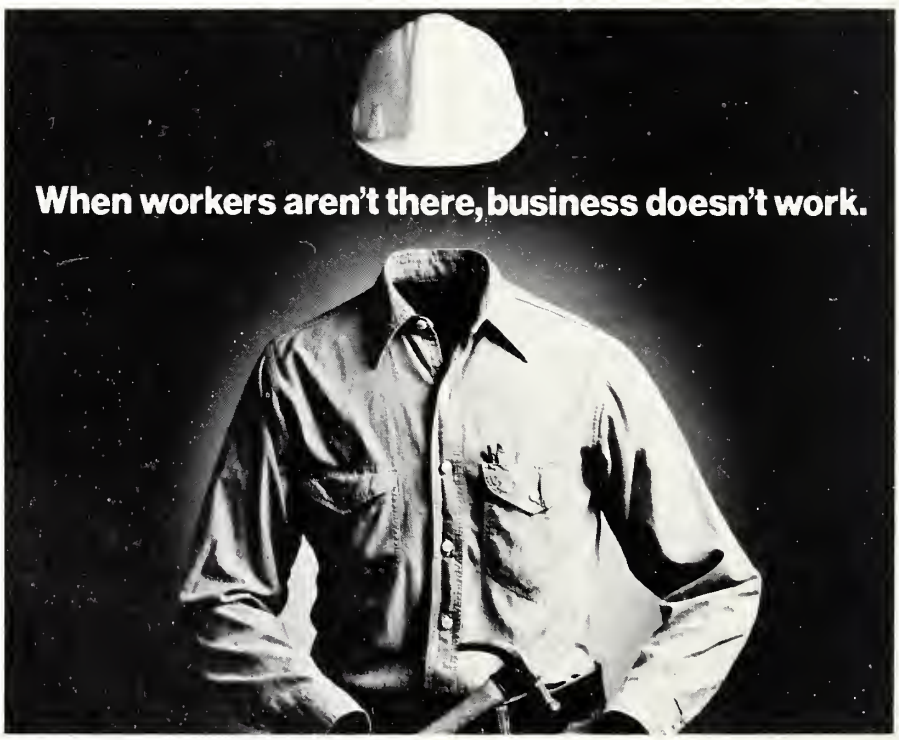

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S
D
Clinicopathologic Conference

Thirty Year Old Caucasian Male With Acute Onset of Abdominal Pain

Mark Odland, M.D.*
Discusser

John F. Barlow, M.D.†
Editor

Case #997 172

This 30 year old Caucasian male worker at Morrell's entered Sioux Valley Hospital because of abdominal pain of one hour's duration.

This patient had been a previously healthy young man with no significant symptoms when he developed suprapubic abdominal pain which radiated into the right upper quadrant and right costovertebral area. In the emergency room, microscopic hematuria (6-7 red cells/hpf) and 4+ glucosuria were found on urinalysis with a 5% dextrose intravenous in place. Blood sugar was 132 mg/dl and blood urea nitrogen and creatinine were normal. White count 12,500/mm^3 (12.5 $\times$ 10^9/L). There was no history of genitourinary symptoms or renal trauma.

PHYSICAL EXAMINATION: Cooperative young man in moderate distress — temperature 102.4°F, pulse 82/min and regular, respirations 22/min and regular, blood pressure 130 systolic and 82 diastolic. There was no abnormality in the head and neck. The lungs were clear to auscultation and percussion. The heart was not enlarged and had a normal rhythm with no murmurs. Abdominal examination revealed no palpable organs or masses. There was mild tenderness in the suprapubic region and punch tenderness in the right costovertebral angle. The testes and external genitalia were normal. The rectal examination demonstrated a minimally tender prostate on the right. The neurologic examination was unremarkable. Pulses in the neck and extremities were normal.

LABORATORY DATA: Urinalysis (multiple studies): amber to yellow, clear, specific gravity 1.023-1.035, pH 5.0-7.0, 2+ ketonuria (on initial specimen only), negative protein, bilirubin, trace glucose (on initial specimen only); hemoglobinuria 3+ on two occasions: sediment 1-4 wbc/hpf, rbc varied from 5-8 to 25-35/hpf. Hemoglobin 15.1 to 13.1 gm/dl, hematocrit 45 to 39 vol/dl; total leukocyte count 4900 to 9200/mm^3 (4.9 to 9.2 $\times$ 10^9/L) with segmented neutrophils 21 to 87% and band neutrophils 18% on admission but 2-3% thereafter (multiple studies). Red cell indices, platelet count and red cell morphology were within normal limits. Blood chemistry results: phosphorus 1.4 mg/dl (normal 2.1-4.5 mg/dl), glucose 153 mg/dl (60-125 mg/dl). Lactic dehydrogenase (LD), alkaline phosphatase, aspartate aminotransferase (AST, SGOT), bilirubin, creatinine, blood urea nitrogen, amylase, potassium, chloride, total CO2 content, uric acid, calcium, total protein, cholesterol were within normal limits. Sodium ranged between 130 to 134 meq/L and glucose was within normal range except for admission values with intravenous glucose running. Prothrombin time and partial thromboplastin times were within normal limits.

IMAGING DATA: Chest film, barium enema, upper gastrointestinal series and liver spleen scan were within normal limits. An oral cholecystogram showed poor visualization of the gallbladder. An ultrasound showed a thickened gallbladder wall. An intravenous pyelogram showed a more medial course than usual of the right ureter near the 5th lumbar vertebra.

HOSPITAL COURSE: On the ninth hospital day the patient spiked a fever to 105.2°F with chills but without localizing pain. He had had a fever of from 101-103°F since admission. The abdominal pain and mild tenderness had shifted from the suprapubic region to the right upper quadrant. The patient had received sulfamethsoxazole, metronidazole, gentamicin and ampicillin. A blood culture grew Bacteroids fragilis subspecies vulgatus with the following susceptibilities at the time of the temperature spike:

	MiC (mdg/ml)	
Carbenicillin	32	susceptible
Cefoxitin	16	intermediate (moderately susceptible)
Chloramphenicol	4	intermediate (moderately susceptible)
Clindamycin	1	intermediate (moderately susceptible)
Metronidazole	0.5	susceptible
Penicillin	4	intermediate (moderately susceptible)
Tetracycline	16	resistant

An operation was performed.

* Resident in Family and Community Medicine, USD Affiliated Program, Sioux Falls, SD
† Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

DR. ODLAND: After being given the presentation of this case, my initial impression was that this man had an acute pyelonephritis of the right kidney. This conclusion changed after the test results were reviewed. The major items which were disturbing in reaching my initial diagnosis were the absence of pyuria and the question of why this otherwise healthy thirty year old man should contract a pyelonephritis. Some conditions which might predispose this man to pyelonephritis are: prostatitis with a subsequent ascending urinary tract infection; an altered immune state accompanying a neoplastic disorder such as a lymphoma, leukemia, or Hodgkin's disease, etc.; decreased resistance secondary to alcohol abuse; or diabetes mellitus, which is a consideration with the reported glycosuria, ketonuria and hyperglycemia. However, intravenous glucose was being given at the time these tests were performed.

A ureteral calculus could account for both the pathogenesis of pyelonephritis as well as the absence of pyuria; but the history is rather atypical for a ureteral calculus.

In addition to probable acute pyelonephritis, the differential diagnosis at this point would have included acute gastroenteritis or colitis, peptic ulcer disease, and gallbladder disease. Another possibility that might easily be overlooked is appendicitis which should be included because of the patient's age and because the presenting complaint is abdominal pain.

I would have begun management with the assumption that I was treating an acute pyelonephritis. This would include obtaining blood cultures, a catheterized urine culture, complete blood count, electrolytes, urinalysis, and 12 panel chemistry and instituting intravenous hydration and ampicillin. Addition of an aminoglycoside would depend on how toxic the patient appeared. Other early studies might have been an intravenous pyelogram and a chest film. The plan would then be to await results before proceeding.

A positive blood culture for Bacterioides fragilis dramatically changed the complexion of this case. Now, an abscess or a suppurative condition from a perforated abdominal viscus comes to mind. This species of bacterium reportedly constitutes greater than 95% of the fecal flora. Bacteroides infections are known to progress rapidly to septicemia. The only other locations where this bacterium is a common inhabitant are in the upper respiratory tract and in the female genital tract. Other important information available at this time are a continued fever of 101-103°F and an intravenous pyelogram showing a medial deviation of the right ureter at the level of the 5th lumbar vertebra.

The differential diagnosis would now be expanded to include colonic diverticulitis, bowel obstruction or Crohn's disease with an intraabdominal abscess. Specific types of abscesses to be considered would be perirenal, periappendiceal, periprostatic and perirectal.

An upper gastrointestinal x-ray series, abdominal sonogram, and barium enema were negative. These negative studies cast doubt on the diagnoses of peptic ulcer disease and Crohn's disease, and eliminate bowel obstruction and diverticulitis as possibilities. The oral cholecystogram shows a poorly visualized gallbladder which has a thickened wall on sonography. The latter is suggestive of chronic cholecystitis but can also be seen when Crohn's disease affects the gallbladder. Laboratory studies do not support an obstructive problem involving the common bile duct.

A perirenal abscess could certainly account for the diversity of findings since it can dissect through the retroperitoneum in any direction. However, it was not seen on the intravenous pyelogram and it could not account for the ureteral deviation at the fifth lumbar vertebral level; perirenal abscess is typically caused by Staphylococcus aureus and is also typically associated with marked leukocytosis.

Another possibility is a prostatic abscess. The literature describes cases of chronic prostatitis becoming acutely symptomatic following prostatic massage or physical examination. Such an acute prostatitis could, of course, result in an ascending urinary tract infection with pyelonephritis. It could also produce a prostatic abscess. This would be an uncommon occurrence in such a young man. The causal bacteria are usually gram negative organisms. Nevertheless, this remains a possibility.

Crohn's disease could account for the variable location of the pain, the thickened gallbladder wall, and could result in a perirectal abscess. In addition the hematuria could result from a rectovesical fistula. A negative past history and a negative barium enema make this an unlikely diagnosis. Also, one should expect associated gastrointestinal symptoms.

The last disease to be considered in my differential diagnosis is acute appendicitis with subsequent appendiceal abscess formation. In an otherwise healthy thirty year old patient with acute onset of abdominal pain, appendicitis must be ruled out. The proximity to the right ureter at the lumbar vertebra 5 could cause medial deviation of the ureter. Any compression of the ureter can result in hematuria. A retrocecal location could account for the initial right costovertebral punch tenderness. This would certainly be an atypical presentation for appendicitis, but would be at the top of my differential prior to surgery.

Regardless of the specific location of the problem, the temperature spike to 105°F and the sudden shift in pain seem ominous, and are suggestive of a ruptured abscess. Exploratory surgery would be indicated.

Concerning management of this case, I would have changed antibiotic accordingly after receiving the antimicrobial susceptibility studies. The only significant thing I would have done differently would have been to order an abdominal computer tomogram after becoming suspicious of an abscess.

Dr. Odland's Diagnosis
Acute Perforated Appendicitis

DR. P. K. ASPAAS:* Was the appendix visualized on the barium enema? In the report on the sonogram of the gallbladder did they comment about the kidneys? The protocol mentions a thickened gallbladder wall, but were there any stones?

DR. BARLOW: There is no mention of the appendix on the barium enema report. There were no stones seen in the gallbladder. No abnormality of the kidneys was noted in the gallbladder sonogram report.

DR. BARLOW: The patient was operated on by Dr. Ellison Kalda.† Dr. Kalda notes in his operative report that he was concerned about the possibility of acalculus cholecystitis because the patient had mild right upper quadrant pain. At surgery the gallbladder was normal and without stones. There was a mass on the right side of the abdomen. This was due to a collection of purulent material elevating the right side of the colon. In dissecting the area, remnants of what were considered to be a necrotic appendix were found and removed. An appendicostomy tube was placed in the right colon and an Axion sump was placed in the area of the abscess. A feeding jejunostomy catheter was also placed as subsequent sepsis was feared. The patient had an uneventful postoperative course and was discharged eleven days postoperatively. This case was indeed a difficult one and the attending physicians considered acalculus cholecystitis as well as many of the conditions described by Dr. Odland.

We received a 4.1 × 1.0 × 0.5 cm fragment of hemorrhagic soft tissue barely recognizable as an appendix. Microscopic sections revealed marked ulceration of the mucosa and gangrene of the appendiceal wall with periappendiceal acute inflammation and abscess formation (Fig. 1).

That acute appendicitis still baffles us is no surprise but it is probably worthwhile discussing this entity. The data I am presenting are all from articles published since 1975. Patient mortality from acute nonperforated appendicitis is approximately 0.1%, but rises to 1% with perforated appendicitis. The latter still constitutes a significant number of cases and is more frequent in patients over 60 and under 1 years of age.

Figure 1
Extensively destroyed appendix with vestiges of remaining mucosa amidst marked acute inflammation.

In a review of 9,652 cases with 26 deaths from appendicitis,[1] ten deaths occurred in cases of nonperforated appendicitis and 16 in cases of perforated appendicitis. This is a fatality rate of six times greater for the latter group. In patients over 50, the signs and symptoms are very similar to the younger age group but perforation occurs during a shorter time interval. 10% of cases of appendicitis occur over the age of 50 and many perforate.

A good reference for modern statistics was published by the Department of Surgery at the San Francisco General Hospital.[2] 1,000 cases of appendicitis were reviewed. The overall negative appendectomy rate was 20% but in women between 20 and 40 years of age the rate exceeded 30%. Two thirds of these explorations negative for appendicitis were performed for nonsurgical conditions most commonly pelvic inflammatory disease or other gynecologic conditions in women. Mesenteric adenitis, gastroenteritis and abdominal pain of unknown etiology accounted for other cases in females and for most of the "normal appendices" in males. The rate of perforated appendicitis was 19% for ages 11 to 30 but 38% for patients less than 10 and 27% for patients over 50 years of age. Abdominal pain and direct tenderness were present in 99% of cases; rectal tenderness in about half of all patients. The

* Medical Director, Family and Community Medicine Program, Sioux Valley Hospital, affiliated with USD School of Medicine and Specialist in Internal Medicine, Sioux Valley Hospital, Sioux Falls, SD.
† General Surgeon, Sioux Valley Hospital and Central Plains Clinic & Faculty, USD School of Medicine, Sioux Falls, SD.

temperature and white count values showed considerable overlap between appendicitis and pelvic inflammatory disease and between perforated and nonperforated appendicitis but were useful in distinguishing appendiceal inflammation from other nonsurgical causes of abdominal pain.

A harder problem to quantitate is the morbidity factor in appendicitis but clearly perforation was associated with a higher wound infection rate.

FINAL ANATOMIC DIAGNOSIS ACUTE PERFORATED APPENDICITIS (RETROCECAL)

* DR. WARREN JONES: Although most patients may have typical or atypical courses with abdominal pain due to appendicitis, I might state that, perhaps due to degenerative changes in the nerves of the gut, some elderly patients may complain of little or no pain at all with acute appendicitis or ruptured acute appendicitis. I have seen two such cases fairly recently.

† DR. BRUCE VOGT: I might say that some patients are quite stoic. I have seen a patient present two weeks after the onset of acute appendicitis with a large abdominal abscess secondary to a perforated appendix.

* Specialist in Internal Medicine, Sioux Valley Hospital; Faculty, USD School of Medicine, Sioux Falls, SD.

† Faculty, Residency Program in Family and Community Medicine, Affiliated with USD School of Medicine, Sioux Falls, SD.

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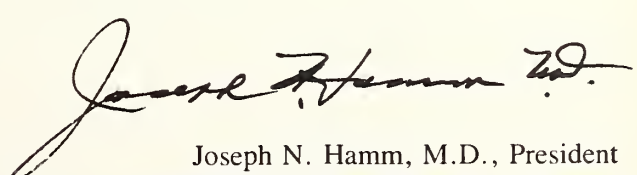
President's Page

The Rostenkowski Amendment was introduced to the United States House of Representatives with little debate. The proposed law required a freeze on the fees of physicians for care of Medicaid patients and mandated assignment of fees. Violation of the freeze was to be punishable by jail, a fine, or both. This part of the amendment was deleted by earlier action of the House, but the assignment was defeated only late in November. Similar legislation will reappear. Chairman of the Senate Finance Committee, Senator Dole of Kansas, has assigned staff to study cost containment measures to conserve the Medicare trust fund. Hearings are planned for early in 1984.

South Dakota physicians are cognizant of the need for cost containment of Medicaid expenditures also. Although physicians' fees are a small part of the cost, the problem has been considered at length. Governor Janklow arranged meetings with concerned health care providers including an ad hoc Committee of the State Medical Association. The committee will report meaningful suggestions for the Governor's consideration. Responsible suggestions to contain cost without deleting essential care for Medicaid beneficiaries have been transmitted to the Department of Social Services.

Physicians cannot control the social problems which escalate the cost of health care. They are concerned with cost containment. We will work with responsible people to try to get the "biggest bang for a buck" for our patients.



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Another Public Health Hazard — The Cordless Telephone

Noise has been established as a cause of inner ear (sensori-neural, SN) type hearing loss in humans and laboratory animals. Noise can be broad spectrum or narrow band in frequency, and may be sustained or intermittent in character. The effect upon the cochlea by noise is contingent upon the sound pressure level in decibels (db), the frequencies involved, the character of the noise, and the total duration of exposure. In individuals who have hearing losses, due to noise or other factors, additional noise exposure has the potential to increase the severity of the disability. For safety in the workplace, or elsewhere, it is usually recommended that the ambient sound pressure level be kept below 90 db.

In the infancy of the telephone industry switchboard operators in rural areas frequently were troubled by annoying noises that accompanied static electrical discharges affecting the telephone lines, especially during electrical storms. Quite often the operators who worked for a number of years developed SN type hearing losses, attributable to the recurrent and prolonged noise exposure. This situation was rectified by the introduction of efficient static discharge arrestors which prevented the sudden surge of electrical energy that manifested itself as noise in headsets.

It appears that the people of the United States have traversed a full 360°, to the place where the telephone is again a factor causing SN type hearing losses.

In a short article that appeared in the current issue of The Bulletin of the American Academy of Otolaryngology — Head and Neck Surgery (AAO-H&NS)[1] it was noted that cordless telephones can cause permanent SN type hearing losses. Cases were documented of SN loss caused by three different brands of cordless telephones that produced sound pressure levels of 141 db in the frequencies 700-1000 Hz (cycles/sec). In view of the fact that the critical speech frequency range encompasses the 500-2000 Hz frequency range, hearing loss in this region has the potential to affect communicative ability.

The AAO-H&NS article noted that the Federal Communications Commission, which certifies telephone equipment, reported that there may be 3-5 million cordless telephones in the U.S., and 30-40 basic models with 150-200 variations. Sales of these units are predicted to rise, projected to one billion dollars in the next two years.

Improper use of the cordless telephones is the usual cause of SN hearing losses. In the conventional wired telephone, ringing stops automatically when the telephone is answered. Cordless units have a flip switch which is usually in the "standby" position, and must be moved to the "talk" position for conversation. If the switch is not moved into the "talk" position, the unit continues to ring directly into the user's ear with sufficient energy that painful sounds and tinnitus may be produced. In addition, radio interference may be a factor producing loud popping sounds in some cordless telephones.

Complaints concerning the SN hearing losses secondary to use of cordless telephones have been filed with the Consumer Product Safety Commission (CPSC) by members of the AAO-H&NS, and the matter is now under investigation. Of the numerous complaints filed with the CPSC, some cases had no hearing loss, but others had hearing losses of 20-25%.

At the behest of the CPSC, warning literature and stickers have been adopted by some cordless telephone manufacturers. A "consumer alert" has been proposed by the CPSC, and the CPSC may work with manufacturers to develop voluntary standards to modify equipment. In the event a "substantial" hazard is established, authority rests in the CPSC to recall cordless telephones. Should this come to pass, the FCC could take cordless telephone equipment off the market by revolking the certification to sell it.

Members of the AAO-H&NS have been alerted to help investigate this situation by collecting case reports and forwarding as many details as possible to the AAO-H&NS. The collected case reports will be directed to the CPSC, accompanied by appropriate recommendations.

Physicians who practice in South Dakota should take note of this situation because patients may be seen, complaining of tinnitus or hearing loss, in whom the cordless telephone may be a factor.

John B. Gregg, M.D.
Willis F. Stanage, M.D.
Lawrence Massa
Office of Medical Services

REFERENCE

1. Cordless telephones can cause permanent hearing loss. **The Bulletin, Am Acad of Otolaryn — Head and Neck Surg.** 2:3, (Oct.) 1983.

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Atrioventricular Block — A Review

Michael R. McVay, M.D.*

ABSTRACT

Atrioventricular conduction involves the transmission of impulses over the specialized conduction system. This specialized conduction system includes the A-V node, the bundle of His, the right and left bundle branches and the Purkinje fibers. The term A-V block is used to indicate impaired conduction of electrical impulses from the atria to the ventricles. Unfortunately, neither the electrical activity of the specialized fibers, nor the site of block is reflected in the standard electrocardiographic leads.[1]

Initially, Wenckebach described two different types of A-V block by inspection of the arterial and venous wave forms.[2, 3] With the invention of the clinical electrocardiogram, A-V block was defined further and in the past 15 years the advent of clinical cardiac electrophysiology with His bundle recordings has given us added information regarding the areas and degrees of A-V conduction delay and block. It is the purpose of this article to review the various types of A-V block, and discuss their electrocardiographic features, electrophysiology, clinical implications and treatment.

ANATOMY AND PHYSIOLOGY OF THE A-V NODE AND HIS PURKINJE SYSTEM

Impulses arising from the sinus node will pass through intra-atrial tracts to reach the A-V node. They then pass into the common Bundle of His and down through the bundle branches into the network of Purkinje fibers of the ventricles.

Transmembrane action potentials recorded from cells within the A-V node (AVN) differ markedly from those recorded in the His Purkinje System (HPS). A-V nodal cells display reduced action potential amplitude, duration, resting potential, conduction velocity, maximum rate of the upstroke (dV/dT), and threshold compared with cells in the HPS.[4]

In the HPS, the fast inward sodium current which is responsible for the rapid upstroke of the cardiac action potential triggers a second slower current carried by calcium ions which is responsible for the plateau phase of the cardiac action potential. Within certain regions of the AVN, it is possible that only the second, slow calcium current may be operative. Cells on either side of the AVN may demonstrate a continuum. These distinctive electrophysiologic differences between normal cells in the AVN and normal cells in the HPS account for differences in the nature of conduction and block. Figure 1 illustrates the action potentials of different regions of the conduction system.

Several different mechanisms may exist which give rise to block in the A-V conduction system. The reader is invited to review this in more detail, in the references that are provided.[4-5]

It should be remembered that block is an abnormal delay or failure of conduction and should be kept distinct from normal delay due to physiological refractoriness of the AVN. Atrial flutter will frequently show 2:1 or 3:1 conduction through the AVN which is a normal phenomenon. Also, one cannot implicate block unless there is adequate opportunity for conduction. For example with an accelerated idioventricular rhythm, one will often see a competing ventricular pacemaker focus over-ride the sinus node, not allowing normal A-V conduction to occur. There is dissociation between the ventricles and atria

* Clinical Assistant Professor of Medicine for the USD School of Medicine in Yankton, SD. He is also a practicing cardiologist at the Yankton Medical Clinic, P.C., Yankton, SD.

but no pathologic block at the AVN. In this instance, the absence of conduction does not signify block.[11]

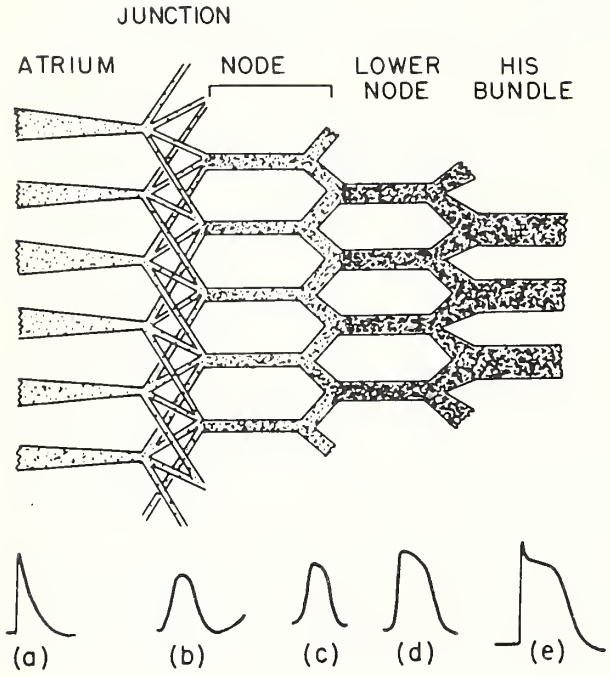

Figure 1
Schematic diagram of A-V region showing transmembrane actions potentials in the atrial (a), atrionodal junctional (b), nodal, and His bundle fibers. Note the slow upstroke and minimal overshoot in the action potential from the junctional cells (b) consistent with this region's slow rate of conduction. Note also that there is spontaneous depolarization in these fibers characteristic of pacemaking ability. The cells from the atria (a), lower A-V node (d), and His bundle (e), record action potentials with more rapid depolarization and absent spontaneous depolarization consistent with faster conduction and lack of pacemaking capability.
"Reproduced with permission from Lipman, B.S., Massie, E., and Kleiger, R.E.: Clinical Scalar Electrocardiography, 6th Edition. Copyright © 1972, by Year Book Medical Publishers Inc., Chicago. Modified from Hoffman, B.F., and Cranefield, P.F.: Electrophysiology of the Heart. [New York: McGraw-Hill Book Co., 1960]."[12]

HIS BUNDLE ELECTROCARDIOGRAPHY

This method will be briefly described to help prepare the reader to understand the intracavitary electrocardiograms which will follow. Transvenous electrodes are placed so that simultaneous recordings of atrial, His bundle and ventricular depolarization can be recorded (Figure 2).

Consequently, different conduction intervals from sinus node to the HPS can be evaluated. The A-H interval refers to the time of conduction from sinus node through the AVN to the onset of the His bundle electrogram (normal = 50-120m sec.). The H-V interval refers to the time it takes to conduct from the His bundle through the HPS (normal 35-45m sec.). Figure 3 illustrates the relationship of the QRS complex to these respective intervals.

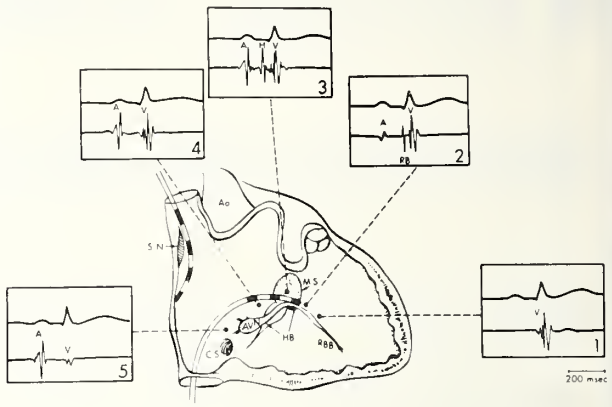

Figure 2
Pull back of the electrode catheter from the right ventricular cavity. A cut away view of the heart demonstrating the electrode catheter lying across the tricuspid valve. The intracavitary electrograms obtained during progressive withdrawal of the catheter are shown in panels 1 to 5. S.N. = sinus node, C.S. = orifice of coronary sinus, AVN = atrioventricular node; HB = His bundle; MS = membranous septum; RB = right bundle branch; A = atrial electrogram; H = His bundle electrogram; V = ventricular electrogram (From Grossman, W.: Cardiac Catheterization and Angiography, Chapter 20. Technique of Recording His Bundle Activity in Man By Gallagher, J.J., Damato, A. with permission.)[13]

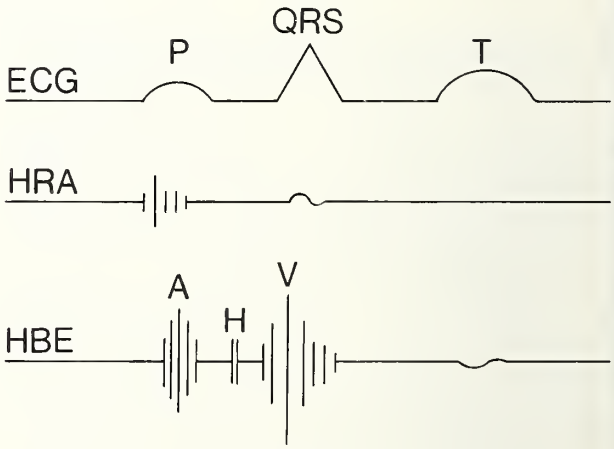

Figure 3
Composite drawing relating the P-QRS-T complex to the His Bundle electrogram. (HRA = high right atrial depolarization electrode; HBE = His Bundle recording electrode; A = Atrial depolarization; H = His depolarization; V = Ventricular depolarization.)

TYPES OF A-V BLOCK

First Degree A-V Block

This is recognized on the surface EKG by a pro-

longed PR interval (>0.21 sec.). This can be a result of conduction delay at one or more sites in the A-V conduction system (atrium, A-V node, bundle of His, bundle-branch-Purkinje network).[6] Evidence exists that the first degree A-V block in a healthy population exists at an incidence rate of 5.2 per thousand.[7] Most commonly, conduction delay is localized to the AVN which is reflected in a prolonged A-H interval. When the QRS complex is narrow, PR prolongation is the result of A-V nodal conduction delay in approximately 85% of patients. In contrast, when the QRS complex is wide, conduction delay is within or distal to the bundle of His in approximately 50% of patients.[6]

Second Degree A-V Block

Mobitz in 1924 first described the electrocardiographic features of second degree A-V block and divided it into 2 types. Type I (Wenckebach) block is characterized by a progressive lengthening of the PR interval preceding a non-conducted P wave (Figure 4). There are other features which usually accompany this phenomena: the greatest increment in delay usually occurs with the second conducted beat of the cycle. The longest PR interval occurs in the beat immediately preceding the blocked P wave and the shortest PR interval is noted in the first conducted P wave after the dropped beat. It is also noted that while the PR interval lengthens in the subsequent beats, the magnitude of the increment diminishes progressively resulting in shorter RR intervals until the P wave is blocked. Conduction ratios of 3:2, or 4:3 are most frequently seen.[6]

The site of block can usually be obtained by the surface EKG. In the presence of normal QRS complexes, the site of Type I second degree A-V block is usually the AVN and rarely within the bundle of His. Type I A-V block within the HPS is usually associated with a pre-existing bundle branch block (BBB) pattern. The increment of PR prolongation is usually, although not always, significantly less than that observed in A-V nodal Wenckebach type of block[8] (Figure 5).

Type II Second Degree A-V Block

Mobitz II block is recognized by a sudden and unexpected block of a P wave without prior prolongation of the PR intervals preceding the dropped beat. In most cases the PR intervals of the conducted beats are normal and the QRS complex shows BBB pattern (Figure 6). In Mobitz II block, the site of block is always within the bundle of His or within the distal HPS (Figure 7). In some cases of Type I, A-V block in the AVN, the increments in the PR interval may be so small that the block may mimic Type II

Figure 4
3:2 Mobitz I (Wenckebach) block showing characteristic gradual PR prolongation until a P wave is not conducted.

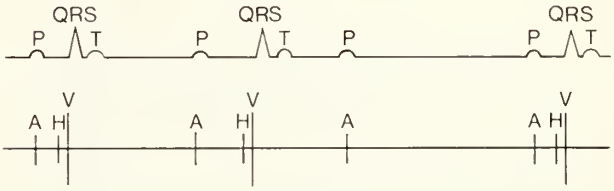

Figure 5
A schematic representation of second degree Mobitz I A-V block with a normal H-V interval. The A-H interval gradually increases until there is a dropped beat indicating the site of block is most likely in the A-V node.

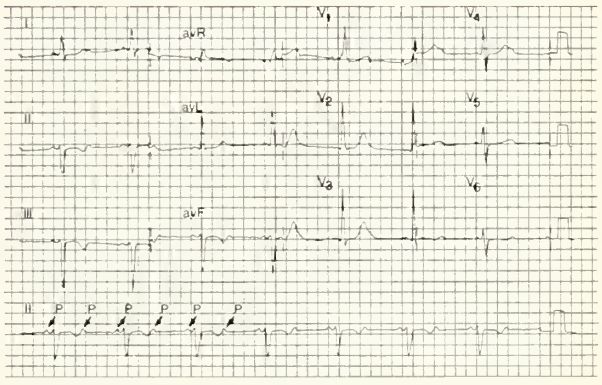

Figure 6
12 lead EKG illustrating Mobitz II A-V block. Note the presence of right bundle branch block and left anterior hemiblock. There is no prolongation of the PR interval before a dropped beat.

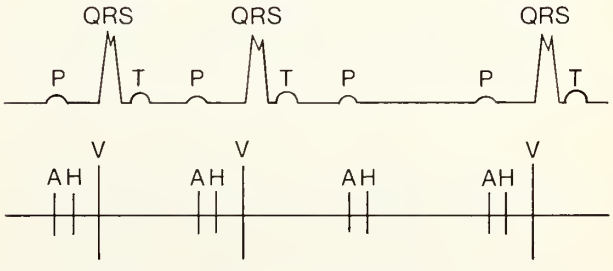

Figure 7
Another schematic representation showing Mobitz II second degree A-V block with a normal A-H interval and a prolonged H-V interval. The block occurs distal to His deflection and bundle branch block is present.

A-V block. However, in these cases a significant shortening of the PR interval, i.e., >20m sec. in the first conducted beat should suggest that the AVN is the site of block.[6]

High Degree (2:1, 3:1) A-V Block

When there is fixed high degree A-V block, the surface EKG is often not helpful in determining the site of block. When the QRS complexes are normal, the site of block is most often within the AVN. High degree A-V block in association with BBB patterns may be within the HPS or AVN.[8] In patients with high degree A-V block, close attention to the PR interval may provide a clue to site of block. A change in the conduction ratio from 2:1 to 3:2 block with a constant PR interval implicates Type II block. A changing PR interval indicates Type I A-V block.[6]

Diagnostic Aids

In some patients with atypical second degree or 2:1 A-V block, it may not be possible to arrive at an accurate diagnosis on the basis of a routine EKG. The following points may be helpful in differentiation and diagnosis:

1. Long rhythm strips may show a change in condition from 2:1 to 3:2 with variation in PR intervals and allow classification into Type I or II block.
2. In some instances with Type I block, the increments in the PR interval may be very slight. The latter changes may not be detectable on the surface EKG at routine paper speed and may mimic Type II block. In these instances recordings at a faster paper speed may enable a more accurate measurement of the PR intervals.
3. The effect of vagal influences is usually limited only to the A-V nodal conduction and not the HPS. Therefore, carotid sinus massage (CSM), if done carefully, may be clinically useful in suggesting the site of block. CSM will usually increase the degree of block when the lesion is in AVN. However, if the lesion is in the HPS, CSM may cause a decrease in the degree of block. Slowing of the sinus rate with vagal stimulation results in improvement to 1:1 A-V conduction and a decrease in block due to a lesser number of impulses arriving at the abnormal HPS. Atropine on the other hand by accelerating the atrial rate may increase A-V block especially through the HPS. Similarly, in patients with atypical patterns of second degree A-V block and fluctuating PR intervals, interventions such as vagal stimulation, atropine, exercise, and atrial pacing may help in classifying second degree block into Type I or Type II.[5]

Third Degree A-V Block

Third degree A-V block refers to the complete absence of A-V conduction. Complete A-V block may be congenital or acquired. In congenital heart block, most frequently the block is in the AVN. In the acquired type, the most frequent site is distal to the bundle of His and represents bilateral BBB. The site of block can usually be predicted from the rate and the morphology of the escape QRS complexes. When the block occurs within the AVN, the escape pacemaker is in the bundle of His and the QRS morphology is identical to that observed during sinus rhythm. Furthermore, the ventricular rate is greater than 40 per minute. This pacemaker will respond to exercise, atropine and isoproterenol by increasing its rate.

When the site of block is in the HPS, the QRS complex shows a BBB pattern and the escape pacemaker rate is usually less than 40 per minute. This focus rarely responds to physiological maneuvers that usually increase heart rate.[8]

A-V Block Due to Concealed His Bundle Extrasystoles

Extra systolic impulses arising from the bundle of His and concealed antegradely within the HPS (no ventricular response) may cause conduction delay or block of subsequent sinus impulses. These concealed extrasystoles may cause first degree, both types of second degree and high degree A-V block. This diagnosis may be made from the routine EKG by using long rhythm strips to look for instances when the extrasystoles do conduct to the ventricles[8] (Figure 8).

The above comments regarding both types of second degree A-V block are summarized in Table 1 and Figure 9.

PROGNOSIS AND TREATMENT

The presence of A-V block often raises the question whether pacemaker implantation is needed for improvement in symptoms or to prevent a catastrophic event. The ease of pacemaker insertion should require us to be more critical of our indications. In this regard, each patient should be evaluated in terms of symptoms, electrocardiographic features and the site and degree of block as thoroughly as possible.

First Degree A-V Block

This is not a contraindication to digitalis, propranolol or calcium channel blocker — but they

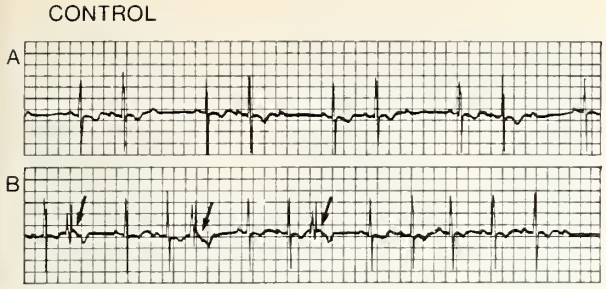

Figure 8

A-V block due to concealed bundle of His extrasystoles. Panel A shows 3:2 A-V block associated with narrow QRS complexes (Lead VI). The P-R intervals of conducted beats are normal and no P-R prolongation is observed prior to the block. Analysis of long rhythm strips (panel B) revealed periodic occurrence of premature QRS complexes with a RBBB pattern just preceding the blocked P waves (arrows). These premature QRS complexes most probably result from aberrant conduction of impulses originating in the region of the bundle of His. Retrograde penetration into the A-V node induces block of the oncoming (panel B) sinus beats (retrograde concealed conduction in the A-V node). When premature His bundle extrasystoles failed to propagate to the ventricles but did propagate retrogradely into the A-V node, the subsequent P waves were suddenly and unexpectedly blocked (panel A). (From Akhtar, M., Damato, A.: Clinical Uses of His Bundle Electrocardiography, Part I, AHJ. 91:520, 1976, by permission.)[8]

TABLE I

SITE OF CONDUCTION BLOCK[6]

	Atrium	AV Node	Bundle of His	Bundle Branch Purkinje Network
1° AV block	Yes	Yes	Yes	Yes
2° AV block				
Type I	Yes	Yes	Yes	Yes
Type II	No	?	Yes	Yes
High degree (2:1, 3:1) AV block	No	Yes	Yes	Yes
Complete AV block	No	Yes	Yes	Yes

should be used with caution. If it is determined that the prolonged PR interval is due to conduction delay in the HPS, then these drugs may be used since they do not affect HPS conduction.

Second Degree A-V Block

All patients who are symptomatic with second degree A-V block should be paced, regardless of the site of conduction delay.[6]

Asymptomatic patients with Type II block or Type I block in the HPS require careful consideration. There have not been any well controlled prospective trials to evaluate which of these patients need pacing. However, clinical experience has suggested that these infranodal blocks are unstable and progress to complete heart block without warning. Prophylactic pacemaker implantation in these patients is therefore justified. Asymptomatic Type I second degree A-V block confined to the AVN — does not require treatment.[5, 6, 9]

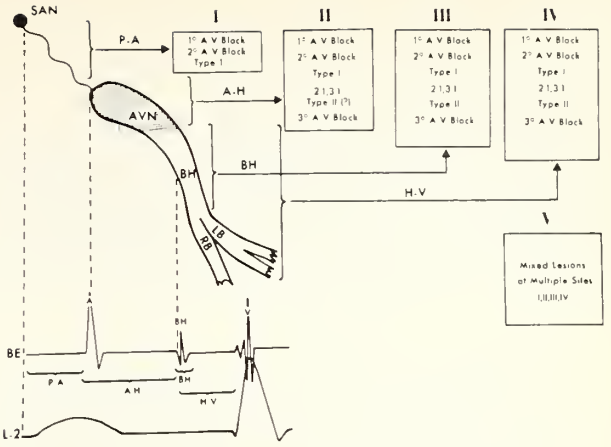

Figure 9

Diagrammatic representation of the A-V conduction system illustrating that the different varieties of the A-V block can occur as a result of lesions in different segments of the conducting system. At the bottom left hand corner are the simultaneous recordings of a standard electrocardiographic lead II (L-2) and a typical bipolar electrogram (BE) from the area of the A-V junction. SAN = sinoatrial node, AVN = atrioventricular node, BH = bundle of His, RB and LB = right and left bundle branches, respectively, P-A = conduction time through the atrium, A-H = conduction time through the AVN, H-V = conduction time through the His-Purkinje system, A = bipolar atrial electrogram, V = the bipolar ventricular electrogram recorded from the area of the A-V junction. (From Narula, O.S.: His Bundle Electrocardiography and Clinical Electrophysiology, Chapter 9. Current Concepts of Atrioventricular Block, by permission).[5]

Inferior wall myocardial infarction may produce second degree or third degree A-V block. Temporary pacing should be carried out if the patient is symptomatic. Because reversible A-V nodal ischemia is the usual mechanism, permanent pacing is rarely indicated.

Anterior wall infarction is often associated with extensive damage to the septum and conduction system. Consequently, the appearance of second degree A-V block can be a harbinger of complete A-V block, and temporary pacing is indicated if this occurs. The presence of Mobitz II block in anterior infarction is an indication for permanent pacing. If Mobitz II A-V block is reversible, controversy exists whether permanent pacing is indicated. It has been shown that the incidence of sudden death in this subgroup of patients is less if permanent pacing is carried out.[10]

Complete Heart Block

Asymptomatic patients with CHB may not require a permanent pacemaker if the block is localized in the AVN and the subsidiary pacemaker accelerates with atropine or exercise. Patients with CHB distal to the bundle of His require permanent pacemaker implantation regardless of presence or absence of symptoms because the rate is not as stable and

Stokes-Adams attacks are more common. All symptomatic patients with CHB require pacing.[5, 6]

Acknowledgement

The author appreciates the helpful assistance and comments of C. Pratap Reddy, M.D., in reviewing this article. Dr. Reddy is Director of the Clinical Electrophysiological Laboratories, University of Kentucky Medical Center, Lexington, Kentucky. Also, I wish to thank Joyce Olson for her technical assistance and M. J. McFadden of Medical Illustrations for her preparation of the figures.

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2. One of the main complaints among patients is the long wait to see their doctor. Try to keep waiting room time to 15-20 minutes. Periodically survey your patients to see how long they are waiting.
3. Send holiday greeting cards to friends, associates and patients.
4. Send letters of thank you to patients and other doctors who refer you patients.
5. Develop a patient questionnaire to help you evaluate your medical practice.

Practice Strategies

6. Provide patient-education brochures on such topics as menopause, breast self-exam, nutrition, breast feeding, hypertension, glaucoma, contraception, ulcer diet, etc. These can be kept in your waiting room or distributed to patients.
7. Develop a first-aid chart, CPR methods and/or instructions on how to help a choking person, for your patients' medical cabinet. Can be used as bill stuffers or given to new patients.
8. Have plenty of time to talk with your patients and answer all the questions that they have.
9. Always answer the following five questions whether they are asked or not:
   A. What's wrong
   B. What caused it
   C. What you're going to do about it
   D. How much the treatment will cost
   E. How long it will take.
10. Follow-up phone calls are excellent ways to show your patients that you care.
11. House calls are practice builders. Don't make them all the time, but occasionally when you know they are needed.
12. Periodically examine the services which you offer and compare them to those that are being offered in other practices. You may want to offer new ones to attract new patients.

Personnel Strategies

13. To a new physician, as important as the quality of care you give is the way you are perceived by your patients.
14. Project yourself on the patient's level. It is important to make the patient feel at ease so that communications can be established. Ask the patient some questions about his work or hobbies to break the ice.
15. The way you dress is important. Dress like a doctor is supposed to dress. Patients don't want to be treated by a physician who looks sloppy.

Outreach Strategies

16. Send letters to new area residents providing names, addresses and phone numbers of family physicians in your community.
17. Meet the other specialists in your area who might be sources of referral.
18. Meet and remember the names of the receptionists in these referral offices. When the doctor is out-of-town or too busy, a simple thing like addressing her by her name when you talk to her may bring some referrals.
19. Provide names of area family physicians in AAA Tourist Guide Book to tourists requiring medical treatment.
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21. A new physician will receive his/her new patients from several sources. He or she should become familiar with these elements:
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Basic Medical Radiation Physics, MGM Grand, Las Vegas, NV, Feb. 23-26. Fee: $500. 50 hrs. AMA Category I credits. Contact: Mary T. Fletcher, Nat'l Instit. for Prof. Education, 4880 River Bend Rd., Boulder, CO 80301. Phone: 800-421-8452.

Ear, Nose & Throat Problems in Primary Care, Sheraton-Ritz, Minneapolis, MN, Feb. 24-25. Contact: CME, Box 293, Mayo Mem. Bldg., 420 Delaware St. SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Stress Management and Human Illness, The Antlers at Lions-Head, Vail, CO, Feb. 25-Mar. 3. Fee: $335. 22.5 hrs. AAFP, Am. Osteo. Assoc. & AMA Category I credits. Contact: Vail Stress Management Conf., P.O. Box 11338, Denver, CO 80211-0338. Phone: 800-525-5810.

Sixth Annual Vail Sports Medicine Conference, Marriott's Mark Resort, Vail, CO, Feb. 25-Mar. 3. Fee: $335. 22 hrs. AAFP & AMA Category I credits. Contact: Vail Sports Medicine Conf., P.O. Box 11338, Denver, CO 80211-0338. Phone: 800-525-5810.

Fourteenth Annual Aspen Radiology Conference, Aspen Instit. for Humanistic Studies, Aspen, CO, Feb. 26-Mar. 2. Fee: $335. 20 hrs. AAFP & AMA Category 1 credits. Contact: Aspen Radio. Conf., P.O. Box 11338, Denver, CO 80211-0338. Phone: 800-525-5810.

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Radiation Physics of Nuclear Medicine, The Sands, Las Vegas, NV, Mar. 22-25. Fee: $500. 50 hrs. AMA Category 1 credits. Contact: Nat'l Instit. for Prof. Education, 4880 River Bend Rd., Boulder, CO 80301. Phone: 800-421-8452.

Colorectal Problems in Primary Care, Hyatt Regency, Minneapolis, MN, Mar. 9-10. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Primary Care and Hand Injuries, Yarrow Holiday Inn, Park City, UT, Mar. 9-10. Fee: $225. 9.5 hrs. AAFP & AMA Category 1 credits. Contact: Ronald Mann, M.D., U. of Utah School of Med., CME, Salt Lake City, UT 84132. Phone: 801-581-3713.

Ninth Annual Vail General Surgery Conference, Marriott's Mark Resort, Vail, CO, Mar. 10-17. Fee: $335. 24 hrs. AAFP & AMA Category 1 credits. Contact: Vail Gen. Surg. Conf., P.O. Box 11338, Denver, CO 80211-0338. Phone: 800-525-5810.

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April

Neuro-Ophthalmology, Holiday Inn Downtown, Minneapolis, MN, Apr. 2-3. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Sexually Transmitted Diseases, Hennepin County Med. Ctr., Minneapolis, MN, Apr. 4. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN. Phone: (612) 373-8012.

Long Term Care, Duluth, MN, Apr. 6. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Electroconvulsive Therapy — 1984 Update, Sheraton Inn, Madison, WI, Apr. 6-7. Fee: $200. 10 hrs. AMA Category I credits. Contact: Dorothy Davidson, Dept. Psychiatry, U. of Wisc. Hosp. & Clinics, 600 Highland Ave., Madison, WI 53792. Phone: (608) 263-6129.

Fifth Annual Topics in Clinical Medicine Symposium, Holiday Inn City Centre, Sioux Falls, SD, Apr. 6-7. Contact: Norma Lubben, Central Plains Cl., 2727 S. Kiwanis Ave., Sioux Falls, SD 57105. Phone: (605) 335-2727.

Pediatric Emergencies, Battenfeld Aud., U. of Kansas, Kansas City, KS, Apr. 9-10. AAFP & AMA Category I credits. Contact: Jan Johnston, Off. of CME, U. of KS Med. Ctr., Rainbow at Olathe Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

Eating Disorders, Earle Brown Ctr., St. Paul Campus of U. of MN, Apr. 13-14. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

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Volume XXXVII February 1984 Number 2

SOUTH DAKOTA JOURNAL OF Medicine

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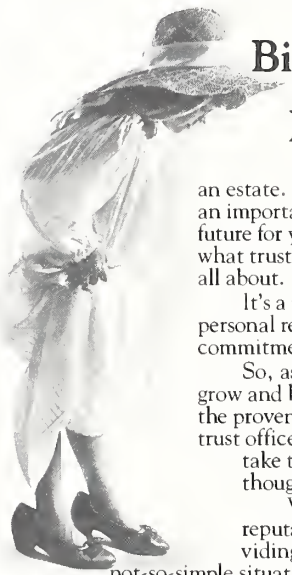

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Volume XXXVII/No. 2 February 1984 (ISSN 0038-3317)

Official Publication of the
South Dakota State
Medical Association

the South Dakota Chapter
of The American Academy
of Family Physicians

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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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Hepatomegaly and Jaundice Occurring Thirty Years After a Subarachnoid Hemorrhage

Douglas M. Traub, M.D.*
Morris L. Radack, M.D.‡

ABSTRACT

A case of hepatocellular hepatoma thirty years after exposure to Thorotrast is presented. The striking laboratory, radiographic and pathologic findings preceding clinical decompensation and death are reviewed. Thorotrast was a radiopaque contrast agent used in many countries prior to 1960. The wide spectrum and latency of neoplastic changes in humans after exposure to this agent are reviewed to emphasize the need for close follow-up in patients with history of exposure.

Research in pharmacology applied to clinical medicine has produced many useful drugs. With this multitude of therapeutic and diagnostic agents comes a seemingly endless list of potential side effects. Unacceptable or life-threatening side effects have been linked with drugs as varied as a long acting non-steroidal anti-inflammatory agent, a uricosuric diuretic, and an oral hypoglycemic agent, leading to their removal from the market.[2, 4, 5]

Side effects in the above mentioned cases became evident very shortly after administration of that particular drug. This report discusses a case of liver disease occurring thirty years after administration of a diagnostic agent used in radiology, Thorotrast.

Case Report

D. J., a sixty-four year old surveyor for an engineering firm, came to the clinic complaining of "feeling run down" for the previous ten days. He complained of food not tasting right and had lost 6.4 kg. in the last several months. Dull generalized fullness in the abdomen had occurred intermittently without nausea or vomiting. Despite years of occasional constipation, no recent changes in bowel habit or stool color had been noticed. Late afternoon feverishness was noticed several days before the clinic visit, but no shaking chills had been experienced.

Medications on admission to the hospital included clonidine hydrochloride, hydrochlorthiazide, and a potassium supplement. Past history was noteworthy for essential hypertension controlled medically for twenty-one years. An episode of left hemiparesis in 1950 was diagnosed as a subarachnoid hemorrhage by cerebral angiography with a radiopaque contrast agent. After an uneventful recovery period, the hemiparesis resolved. An episode of chest pain in 1972 was ascribed to angina pectoris following a graded exercise test described as showing "borderline ischemia." A routine chest x-ray in 1976

* Third year Internal Medicine Resident, Department of Internal Medicine, USD Affiliated Hospitals Internal Medicine Residency Program, Yankton, SD.
‡ Clinical Associate Professor, Department of Internal Medicine, USD Affiliated Hospitals Internal Medicine Residency Program, Yankton, SD.

showed several small calcified granulomas, but a skin test with Histoplasma antigens was unreactive.

The patient had smoked cigarettes for ten years in the distant past. He consumed 1-2 beers daily but used no illicit drugs. There was no past exposure to benzene or other industrial toxins.

The physical examination found an icteric but well developed man appearing to be in his early sixties. His pulse was 76 per minute (regular), temperature 98.8°F, respirations 20 per minute, and the blood pressure 148/96 mmHg (sitting). He weighed 181 lbs. (82.3 kg.). Scleral icterus (mild) was present on the head and neck examination. The heart and lungs were normal. A firm, tender liver edge descended six cm. below the right costal margin, but no nodularity was described. Percussable liver dullness spanned 15 cm. No splenomegaly was present, and the remainder of the abdominal examination was unremarkable. There was no peripheral edema. On neurologic evaluation, a mild left central seventh nerve deficit was evident, but the remainder of the examination was normal.

The admission laboratory data revealed: (normals in parentheses) Aspartate aminotransferase (AST/SGOT) 77 IU/L (8-30), lactic dehydrogenase (LD) 245 IU/L (50-193), alkaline phosphatase 431 IU/L (36-92), and the total bilirubin 1.8 mg/dl (0.1-1.2). The hemogram was as follows: hemoglobin 13.7 gm/dl, hematocrit 42 vol/dl, WBC 15,300 mm^3 (15.3 $\times$ 10^9/L) with 73% segmented neutrophils, 18% lymphocytes, 7% monocytes, and 2% eosinophils. Platelets numbered 438,000 mm^3 (438 $\times$ 10^9/L) while the sedimentation rate was 103 mm per hour (Westergren). The urinalysis was normal. Serum electrolytes included sodium 133mEq/l, potassium 3.9 mEq/l, and the chloride 96 mEq/l. Screens for viral hepatitis were negative including core antibody (anti HB_c) and surface antigen for hepatitis B (HB_sag), and antibodies to hepatitis A (IgM anti HAV). The prothrombin time was 13 secs. with a control of 12 secs. An assay for gamma glutamyl transpeptidase (GGTP) was elevated four times normal at 263 units.

A technetium sulfur colloid liver/spleen scan displayed a mildly enlarged liver with multiple irregular focal defects. The spleen was not visualized but increased uptake by the bone marrow was noted, compatible with mild liver dysfunction. An intravenous pyelogram was normal except for extensive radiodense areas in the spleen and the right upper abdomen adjacent to vertebrae T-11 and T-12 (Figure 1). A chest x-ray showed only multiple small calcified granulomas. Collections of radiodense material in the liver and a densely opacified spleen were identified on a computer tomogram (CT) of the abdomen (Figure 2).

Figure 1
Intravenous pyelogram with densities in the liver, spleen and nearby lymph nodes.

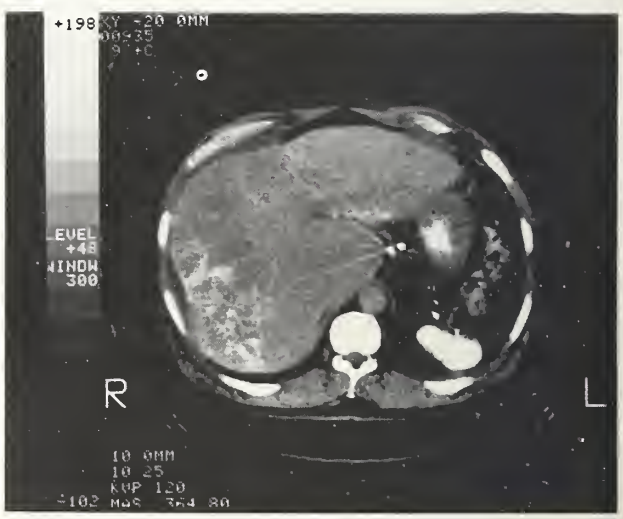

Figure 2
CT scan of upper abdomen with radiodense deposits in the liver and spleen.

The following studies were drawn but later reported negative by a reference laboratory: Cryptococcal antigens, and antibodies for Histoplasmosis, Blastomycosis, and Coccidioidomycosis by complement fixation. A percutaneous needle biopsy of the

liver yielded a core of tissue displaying cord-like fibrosis of the liver with peculiar deposits of yellow-brown pigment (Figure 3).

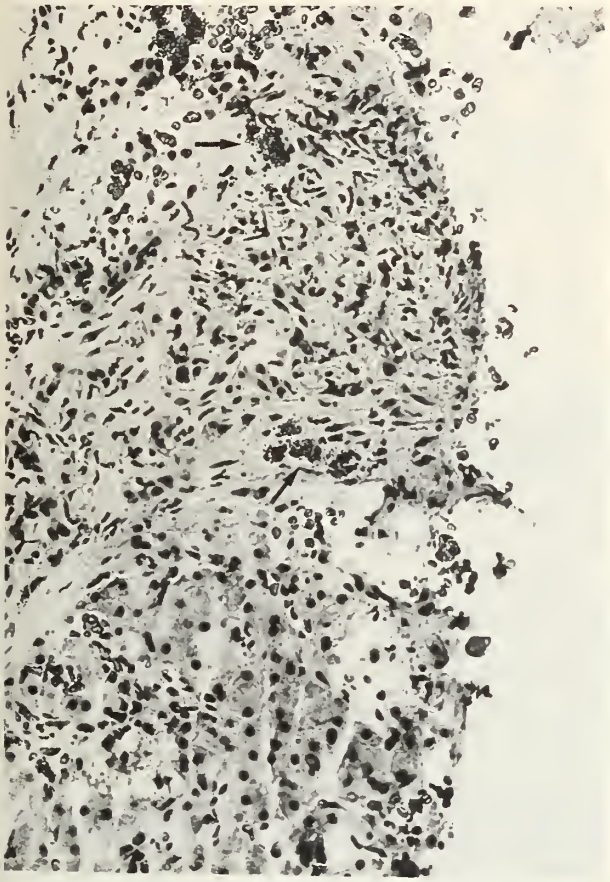

Figure 3
Fibrosis, early cirrhosis, and dark colored granules seen on needle biopsy of the liver. (x40)

The patient was then referred to the same medical center at which he was evaluated for subarachnoid hemorrhage years prior. A review of medical records there, confirmed that the patient had received 30 ml of Thorotrast as part of a radiologic study to detect intracranial bleeding. Further studies at the referral center completed 1/83 included a CT directed needle biopsy of the liver which obtained a core of tissue positive for grade III adenocarcinoma compatible with a hepatocellular primary.

The patient was offered chemotherapy but he declined and was discharged on cholestyramine to control itching. Rapid hepatic decompensation ensued and the patient died four months later. At autopsy, a large hepatoma was present with ascites and peritoneal adhesions. The liver weighed 3,990 grams and had a roughened, opaque capsule diffusely adherent to the dome of the diaphragm. The parenchyma on section was red-tan but contained innumerable yellow-tan nodules up to six centimeters in diameter (Figure 4). Greatest involvement with the nodules was noted in the right lobe where many nodules coalesced with each other to nearly obliterate the intervening bile stained parenchyma. Gross inspection of the pancreas, lungs, stomach, large and small bowel, breasts and gallbladder was negative for other sites of primary tumor.

Microscopic examination of the liver sections found the usual architecture distorted by broad bands of dense fibrous connective tissue containing collections of bile ducts and small foci of hemorrhage. The connective tissue bands separated and surrounded proliferations of neoplastic epithelial cells which resembled hepatocytes. The neoplastic cells formed cords and nests and were seen to contain abundant cytoplasm. Final anatomical diagnosis was hepatoma, hepatocellular type.

Figure 4
Section of liver at autopsy showing massive replacement by tumor.

Discussion

Thorotrast was a radiopaque agent used to image the brain and liver, as well as a contrast material for sinographic studies from the late 1920's to the mid 1950's. Abbatt summarized the many uses of Thorotrast well when he said, "no cavity or orifice remained unknown and unexplored by Thorotrast."[1]

This agent consisted of a colloidal solution of thorium dioxide, containing the isotope radiothorium. Thorium decays predominately as an alpha particle emitter having a half life of approximately 1.4×10^{10} years.[6] In vivo, an intravascular injection of Thorotrast is deposited mainly in the reticuloendothelial system. Sixty percent of the radioactivity accumulates in the liver with small amounts being deposited in the spleen, bone marrow, and

lymph nodes. The biologic half-life in humans is four hundred years.[6]

Scattered reports of local fibrosis, granuloma formation, and liver disease surfaced several years after the introduction of Thorotrast, prompting a report from the International Atomic Energy Commission in 1964. Since that time, follow-up of patients exposed to Thorotrast have confirmed unusually high rates of leukemia, aplastic anemia, bone and hepatic tumors.[3] Reports indicate that hemangioendotheliomas, cholangiocarcinomas, and hepatocellular carcinomas account for most of the liver malignancies discovered. Chromosome studies have confirmed increased chromosomal aberrations in circulating lymphocytes after exposure to Thorotrast.[6]

Worldwide, more than ten thousand patients have been exposed to this agent in its use as a contrast material. The latent interval between exposure and the development of blood dyscrasias or hepatic tumors is eighteen to thirty years.[7] This latency points out the importance of close follow-up for patients with prior Thorotrast exposure.

Summary

A case of Thorotrast associated hepatic fibrosis and hepatocellular carcinoma is presented to review the clinical, radiographic and histologic findings. The need for close follow-up of Thorotrast individuals is also emphasized.

ACKNOWLEDGEMENTS

Thanks to Joyce Olson and Mary Jean McFadden for their assistance in preparing this report and to Thomas Johnson, M.D. for preparing the photomicrographs.

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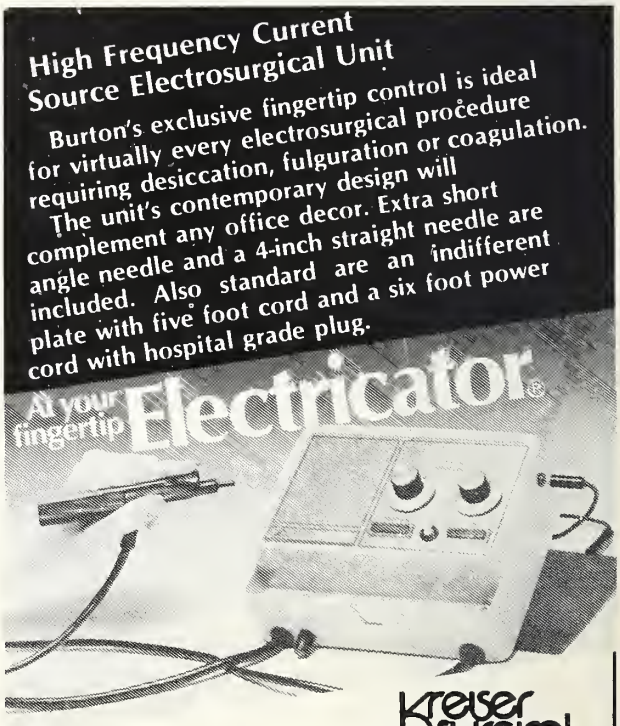

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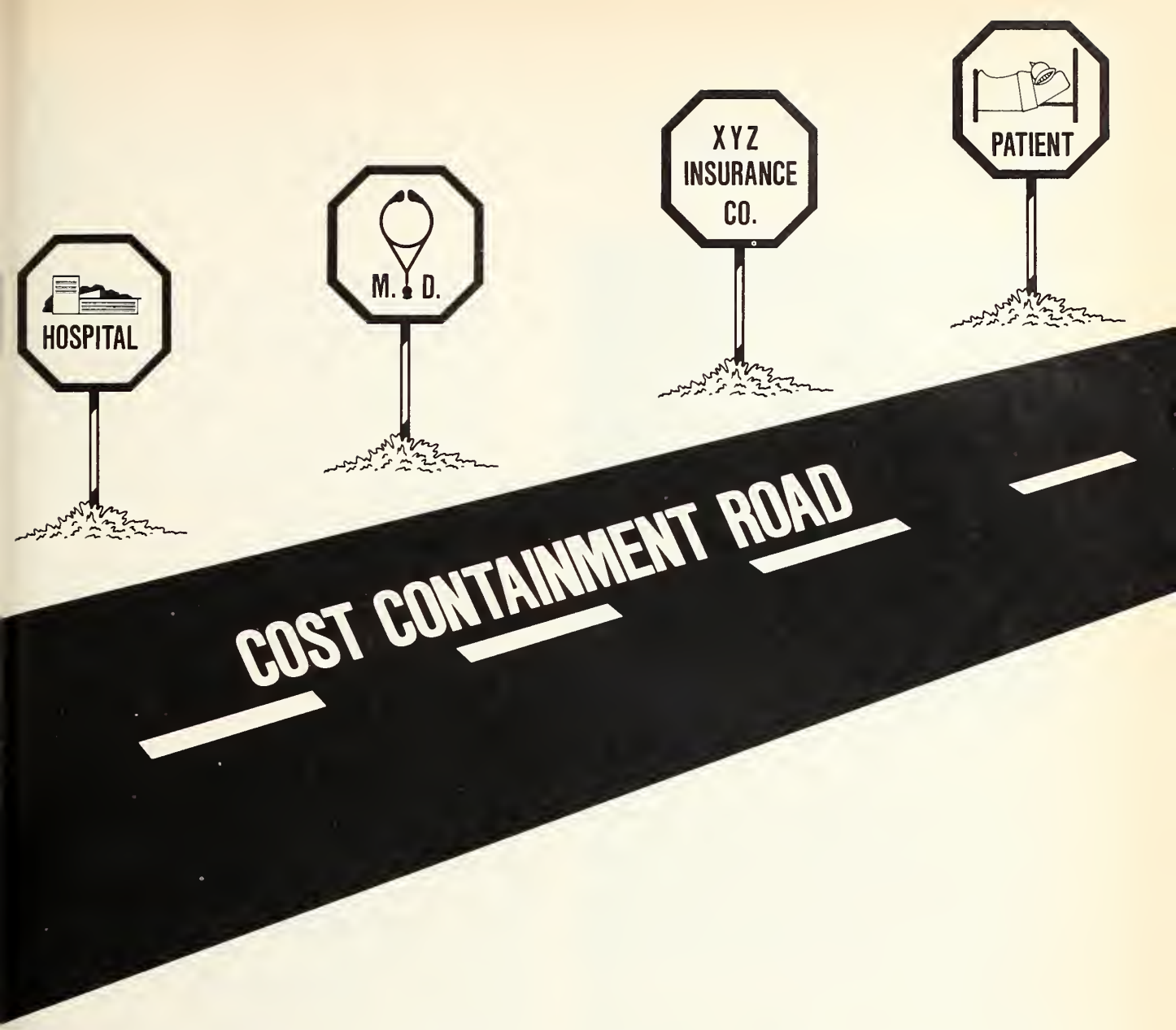

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SD *Council Meeting Highlights*

The Council of the South Dakota State Medical Association met on Friday, November 18, 1983, in Sioux Falls, South Dakota. Following are items of business transacted at this meeting.

1. RISK MANAGEMENT PROGRAM. The Council approved the recommendation of the Commission on Professional Liability to have a risk management session, including a mock trial, in conjunction with the 1984 Annual Meeting provided those in attendance who are insured by the St. Paul Companies would be eligible for a 10 percent premium reduction as last year. Also, the Council approved an ongoing risk management program consisting of a newsletter to members or a page in the *Journal* which would include timely information and tips provided by the St. Paul Companies and the Association's legal counsel.
2. COST CONTAINMENT. The Council considered a report from the Commission on Medical Service's Subcommittee on Cost Containment with regard to instituting meetings with business leaders and physicians to discuss mutual problems concerning health care costs. The Council felt strongly that each district should use its own discretion in instituting such meetings according to their area's needs, and that the State Medical Association would provide assistance to any district if needed.
3. 1984 LEGISLATIVE ISSUES. The State Medical Association will sponsor legislation concerning the coroner's statute, and will endorse legislation on the following: 1) child passenger safety, 2) repeal of the mandatory premarital serology testing, 3) repeal of the annual TB testing for school teachers, and will oppose legislation for the licensure of radiologic technologists. The Council decided the SDSMA should remain neutral regarding the licensure of athletic trainers, they tabled discussion on a proposal to raise the drinking age to 21, they decided not to introduce legislation concerning practice parameters for nurse midwives, and they decided not to co-sponsor legislation requiring CPR certification for high school graduation but rather endorse the concept of CPR training in high schools and provide educational assistance if needed.
4. COUNCILOR FROM MITCHELL DISTRICT. Charles D. Monson, M.D., was named Councilor from the Mitchell District to complete the unexpired term of Richard Gere, M.D. who was elected Vice President of the SDSMA.
5. SPEAKER OF THE HOUSE OF DELEGATES. Frank Messner, M.D., Yankton, was elected Speaker of the House to complete the unexpired term of A. J. Barrett, M.D. who died recently.
6. HONORARY LIFE MEMBERSHIP. The Council elected C. L. Vogele, M.D., Aberdeen, to honorary life member status.
7. CHELATION THERAPY. The Council requested that the South Dakota State Board of Medical and Osteopathic Examiners make a declaratory ruling regarding the medical problems for which chelation therapy has been proven to be efficacious.

■

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President's Page

The most contentious issue to face the House of Delegates of the American Medical Association convened in Los Angeles on December 4-6, 1983, was the composition of hospital medical staffs. State laws assure access to the hospital by limited license practitioners, violations would provoke antitrust prosecution. Conscious of these facts, as well as the responsibility of fully licensed physicians to assure uncompromised quality of care in the hospital, the House adopted an Alabama resolution. The resolution made medical staff policy an issue for the local hospital. Once adopted it became AMA policy, stating that hospital admitting privileges should be granted "in accordance with state law, and in accordance with the criteria for standards of medical care established by the hospital individual medical staff."

The policy was proposed as an amendment to the language for the Medical Staff Chapter of the Accreditation Manual for Hospitals. The amendment was adopted by the JCAH. Under the new standards any practitioner who provides independent patient care must have delineated privileges subject to the rules and bylaws of the medical staff and departments, as well as to quality assurance review. Patients admitted by non-physicians will undergo prompt medical evaluation by a physician with an unrestricted license.

It is our professional responsibility to ensure compliance with the law without compromising the quality of medical care.

Joseph N. Hamm, M.D., President
South Dakota State Medical Association

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S
D
Clinicopathologic Conference

Twenty-Nine Year Old Male With Recurrent Left Upper Lobe Pneumonia

Stephen H. Williams, M.D.*
Discusser

John F. Barlow, M.D.†
Editor

Case # 1000 159

This 29 year old Caucasian farmer was admitted to Sioux Valley Hospital because of a persistent left upper lobe infiltrate in the lung.

Four months prior to admission the patient developed fever between 102-103°F, shaking chills and drenching night sweats associated with cough which produced a variable amount of white sputum without blood. A chest x-ray showed a left upper lobe infiltrate and a sputum culture grew Haemophilus influenzae. The patient was treated with antibiotics of unknown type. A repeat chest x-ray showed some resolution of the upper lobe infiltrate but a mass near the aortic arch was noted. This was thought to be consistent with periaortic lymph nodes. Two months after the first episode the patient had recurrence of chills, fever, night sweats and cough with a sputum culture positive again for Haemophilus influenzae. The patient was treated with doxycycline. Symptoms abated but ten days later the symptoms recurred and the patient was switched to cefaclor. Symptoms again abated but within a week the patient had recurrent symptoms, this time with an episode of bright red blood in the sputum and diffuse left anterior chest pain which was worse on inspiration. There was a mild progressive dyspnea and some palpitations but the patient was still able to engage in active exercise such as basketball. There was some wheezing later in the course but no orthopnea. There was mild anorexia but no significant weight loss. There was no history of travel outside the midwest or exposure to birds. The patient was a non-smoker. The remainder of the review of systems revealed no arthritis, rash, genitourinary or gastrointestinal symptoms.

The patient had had a ruptured appendix nine years prior to admission. There was no history of excess intake of alcohol, epilepsy, drug abuse or aspiration of a foreign body.

PHYSICAL EXAMINATION: Height 6′7″, weight: 98.5 kg., temperature 37°C, respiratory rate 20/min. and regular, pulse 72/min and regular, blood pressure 132 systolic and 60 diastolic. Examination of the head and neck was unremarkable. There was slight deviation of the nasal septum to the left. There were several 1 cm palpable, freely-moveable, nontender supraclavicular lymph nodes. The heart was within normal limits of size with a normal rhythm and no murmurs. The examination of the chest was clear to auscultation and percussion. Examination of the abdomen showed no spasm, tenderness, or palpable organs or masses. There was an old right lower quadrant appendectomy scar. Rectal examination was unremarkable. The pulses in the extremity were 3-4 + and symmetrical. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis yellow, clear, specific gravity 1.019, pH 5.0, negative for protein, glucose, ketones, bilirubin, and hemoglobin; sediment negative. Blood hemoglobin 12.7 gm/dl, hematocrit 38 vol/dl with normal red cell indices, total leukocyte count 6500/mm^3 (6.5 × 10^9/L) with 55% segmented neutrophils, 4% neutrophilic bands, 32% normal lymphocytes, 4% monocytes, 3% eosinophils and 2% basophils. Platelet count was 290,000/mm^3 (290 × 10^9/L). Erythrocyte sedimentation rate was 2 mm/hr. The prothrombin time and partial thromboplastin time were within normal limits. A 12-panel chemistry including lactic dehydrogenase (LD), alkaline phosphatase, asparatate aminotransferase (SGOT, AST), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, cholesterol, and serum protein electrophoresis were within normal limits except for a slightly low alpha I globulin (0.1 gm/dl). Total hemolytic complement was 61 units per liter (borderline low). C3 complement was within normal limits. Febrile agglutinin test for serum antibodies to brucella and tularemia were negative. Cultures of sputum for mycobacteria and fungus were negative. Three blood cultures were negative. Sputum and bronchial washings showed many segmented neutrophils on a gram stained smear with gram positive cocci and gram positive bacilli but no pathogenic organisms were cultured. A PPD

* Resident in Family and Community Medicine, Sioux Falls Program, Affiliated with USD School of Medicine, Sioux Falls, SD.
† Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital. Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

(tuberculin intermediate mantoux) skin test was negative. Serologic tests in serum for antibodies to Aspergillus, Blastomyces dermatitidis, Coccidioides immitis, Histoplasma capsulatum, Toxoplasma gondii, Epstein Barr and Cytomegalovirus were negative. Pulmonary and bronchial washing cytologies were negative for malignant cells.

Review of outside films showed a chest film three months prior to any symptoms to be without abnormality. At the onset of symptoms there was a dense infiltrate in the left upper lobe and a mass above the left hilum was considered a possibility. Films taken in the hospital showed a fairly dense infiltrate just superior to the left hilum which was suggestive of a mass. A bronchoscopy showed a mass in the apical and posterior segment of the left upper lobe. Biopsy showed acute and chronic inflammation but no evidence of malignancy. The patient was subsequently admitted for definitive surgery.

DR. WILLIAMS: According to the history as given in the protocol, the most likely cause of this clinical picture is a nonmalignant tumor of the left upper lobe which is causing bronchial obstruction and recurrent pneumonia. I will list some nonmalignant conditions which may or may not cause obstructing bronchial lesions, but may cause a mass with infiltrate:

Very Common

1. granulomas — histoplasmoma, tuberculoma, coccidioidoma, cryptococcoma
2. other disease — North American blastomycosis and actinomycosis

Common

1. lung abscess — before evacuation into bronchus
2. slowly resolving circumscribed pneumonia of any cause
3. lipoid pneumonia
4. hamartoma

Less Common

1. bronchogenic cyst
2. pulmonary infarction
3. bronchial adenoma
4. arteriovenous fistula
5. enlarged pulmonary artery
6. infected fluid filled bulla
7. rheumatoid nodule

Rare

1. neurogenic tumor such as Schwannoma (neurilemmoma)
2. sclerosing hemangioma
3. lipoma
4. granular cell tumor

Assuming that the laboratory data do not mislead us with false negative results, one can rule out histoplasmoma, tuberculoma, coccidioidoma, cryptococcoma and blastomyocosis although the last may have negative serologic tests. The agent causing actinomycosis, Actinomycis israeli, is really a strict anaerobe. In addition to strict anaerobic conditions, it requires at least 4-6 days before visible growth is noted. This organism is part of the normal flora of the oral cavity and can cause pulmonary infection secondary to aspiration. Commonly this diagnosis is not made until after surgical resection. However, treatment with penicillin for up to 6 months is frequently effective if the diagnosis is made by aspiration.

Aspiration of material from the oral cavity is the major cause of lung abscess and is associated with fever, chills, cough and production of sputum. It should be noted that aspiration of liquid gastric contents usually results in bronchospasm, inflammatory pneumonia and pulmonary edema with a diffuse inflammatory process without necessarily lung abscess formation. The usual causes of aspiration pneumona or pulmonary abscess are anaerobic organisms which are indigenous to the oral cavity. This patient's symptoms are consistent with aspiration, but he had no history of alcoholism, epilepsy or anesthesia to suggest aspiration. One might have attempted to obtain material for culture at the time of bronchoscopy to isolate the organisms causing the disease.

A slowly resolving circumscribed pneumonia can present as a mass like lesion and produce the above picture.

Lipoid pneumonia is a diagnosis rarely made before thoracotomy without a history of mineral oil ingestion or the use of oily nose drops.

Benign hamartomas of the lung are usually intrapulmonary but endobronchial hamartomas may occur. This lesion is a developmental malformation composed usually of cartilage, fat, epithelium and smooth muscle. They most commonly occur in the sixth decade and do not undergo malignant change. Endobronchial hamartoma could produce this picture, but does not usually cause bronchial obstruction. Bronchogenic cyst is a congenital malformation in which portions of the lung have anomalous venous drainage, but there are, of course, cystic changes. They may be unilateral or bilateral and associated with hemoptysis.

Pulmonary infarction may create a mass like shadow, but is often associated with a history of phlebitis or pleuritic chest pain and hemoptysis. It certainly does not cause recurrent pneumonia from bronchial obstruction.

Bronchial adenomas are of three distinct varieties: Carcinoid, adenoid cystic and mucoepidermoid. These were once classified as benign, but they can infiltrate locally and commonly metastasize to regional lymph nodes and hence are better described as malignant lesions. They certainly can produce the picture of obstruction with recurrent pneumonia and are a strong possibility in this case.

An arteriovenous fistula may appear as a pulmo-

nary mass. This lesion may be either a congenital anomaly or acquired secondary to trauma. If large and associated with significant shunting, clubbing, cyanoses and a murmur over the chest may be noted. In the congenital form of arteriovenous malformation, other organs may be involved. This is an unlikely possibility in this case and there is no history of trauma to suggest an acquired lesion.

An enlarged pulmonary artery may be mistaken for a pulmonary mass, but the history of the previous normal x-ray would rule this out. This entity most commonly occurs in patients with congestive heart failure and resolves with the treatment of the heart failure.

Infected fluid in a bulla occurs in individuals with chronic obstructive pulmonary disease, but can occur in healthy young adults. A rheumatoid nodule can produce an intrapulmonary mass, but we have no evidence of either rheumatoid arthritis and endobronchial involvement would be unusual.

This could be any of the benign tumors that I have described above. My major differential diagnosis is between a bronchogenic cyst and a benign intrabronchial neoplasm.

Figure 1
Chest film showing dense infiltrate and mass above lateral and posterior to the aortic knob.

Dr. Williams Diagnosis
Bronchogenic Cyst or
Benign Endobronchial Tumor

DR. HEINEMANN:* May I see the x-ray?

DR. BARLOW: The previous normal x-ray and the developing mass like lesion and infiltrate in the left upper lobe are demonstrated (Fig. 1).

The specimen received was the upper lobe of the left lung. Within the lumen of the apical posterior segmental bronchus was a 2.0 × 1.0 cm polypoid smooth firm mass (Fig. 2). Although tumor protruded into the lumen, the attachment was below the resected portion of bronchus. Microscopically the tumor was composed of dilated glands filled with mucus and lined by flattened or tall columnar mucus secreting epithelium. In other areas, pavement like cells in nests characterized by small uniform nuclei and a moderate amount of eosinophilic cytoplasm and defined cell borders were present (Fig. 3). Mucus secreting cells were seen among these nests. No necrosis on mitotic activity was seen. There was minimal infiltration of tumor at the base of the lesion but the tumor was not encapsulated. The remainder of the lung showed prominent slightly dilated bronchi filled with mucus. There was an inflammatory

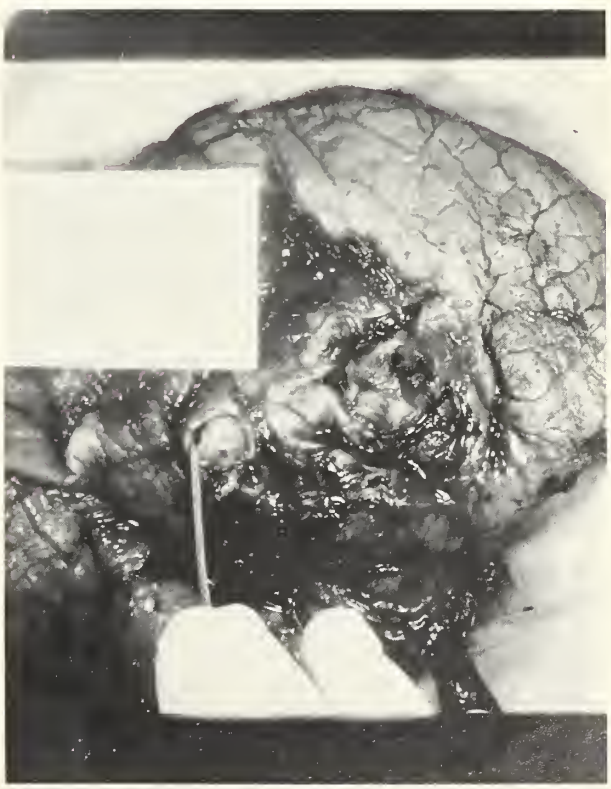

Figure 2
Smooth glistening intrabronchial mass.

* Resident in Family and Community Medicine, Sioux Falls, Affiliated with School of Medicine, University of South Dakota.

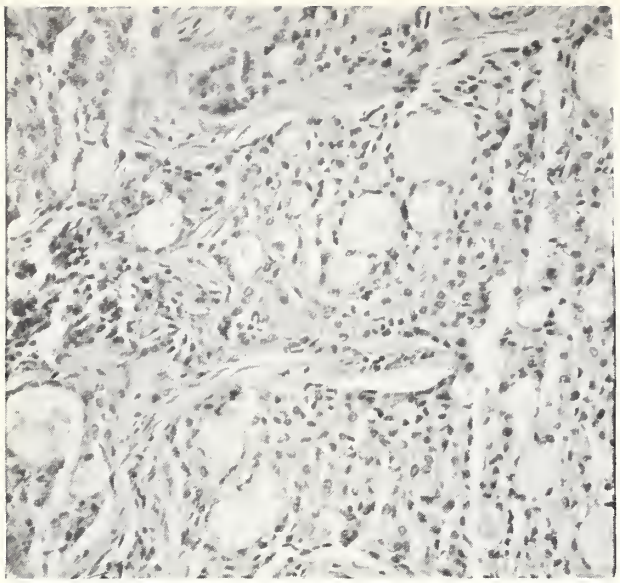

Figure 3
Dilated glands filled with mucus and nests of pavement like epithelial cells typical of mucoepidermoid tumor.

infiltrate around the bronchus ranging from acute pneumonitis to organizing pneumonia. The changes in the lung parenchyma were secondary to bronchial obstruction and infection.

FINAL ANATOMIC DIAGNOSIS MUCOEPIDERMOID TUMOR, LOW GRADE

Mucoepidermoid tumors are the rarest of the so called bronchial adenomas. Leonardi et al collected 7 cases (6 low grade; one high grade) from series of 116 bronchial adenomas and 4250 bronchial carcinomas. Two arose from the trachea and five from a major bronchus. Carcinoid bronchial adenomas are the most frequent and constitute over 80% of the lesions in some series. The next most frequent is adenoid cystic carcinoma (cylindroma) and the least common is the mucoepidermoid tumor constituting 1-5% of bronchial adenomas.

The term bronchial adenoma itself is a misnomer as all these lesions can be locally aggressive or metastasize. Generally, however, they have an excellent prognosis after pulmonary resection.

Mucoepidermoid tumor of the lung was first described in 1952 by Smetana and is identical by light and electron microscopy to its counterpart in the major and minor salivary glands. It arises from the submucosal bronchial glands of the major bronchi and trachea and can show a spectrum of well differentiated to atypical cellular patterns. The well differentiated or low grade lesions such as demonstrated in this case have an excellent prognosis but higher grade lesions may recur or metastasize. At the upper end of the scale high grade mucoepidermoid lesions seem to merge with adenosquamous bronchial carcinomas which have usually a dismal outcome. It should, however, be emphasized that even low grade mucoepidermoid tumors must be considered as low grade malignancies.

The lesion has been described in patients from ages 6 to 76 with two thirds occurring in the 45-70 year age group. There is no relationship to smoking and males with this tumor outnumber females in a ratio of 2 to 1. The endobronchial location of the lesion explains the presenting signs and symptoms which are due to bronchial obstruction. These include cough, hemoptysis, wheezing, recurrent episodes of pneumonia with fever, malaise and weight loss. In all respects, the young man we are discussing was a typical case.

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THE COMPETITIVE EDGE

OUTREACH TO PATIENTS

Marketing Glossary

Communications — The umbrella term for the process of relaying information from person to person, or group to group through various means of transmittal. From Swiss yodeling to the most sophisticated satellite transmission, and everything in between, both verbal and non-verbal. All communications in order to qualify as such must complete a loop — it must be received as well as transmitted. In the aggregate, as communications in the plural, it comprises and encompasses all forms of communicating. Simplest form: I tell you information. You hear and understand my information, and respond in some manner.

Marketing — An ongoing business/professional activity consisting of two aspects (1) determining realistically your own professional needs and your patients' health and human needs, in the aggregate and in detail, and (2) determining the most reasonable ways of meeting those needs and then pursuing them. It is a continuing process and as important to your practice as quality medical training and continuing education, which is the most important thing you need to learn about it. Once you accept that it is important, everything else will follow.

Advertising — The business activity involving purchase of space or time in the public media through which to convey a message of one's own choosing. Advertising properly involves (1) purchase of space or time, and (2) words and/or illustrations of the purchaser's choice; however, it frequently is misused generically to mean any method of favorably promoting a private interest. It long has been considered unethical for physicians to advertise their services, except within tightly controlled circumstances such as new practice announcements and listing in telephone Yellow Pages. In the late 1970s, the U.S. Federal Trade Commission succeeded in forcing professional organizations to liberalize their policies toward advertising by individual lawyers, physicians, etc. This could portend fundamental changes in prevailing attitudes, both among professionals and their clients or patients, toward advertising of individual services, talents and even prices charged for services. The medical profession has not embraced unbridled advertising by physicians, but actively has sought to measure public acceptance of it.

Public Relations — An ongoing activity in which a business or professional person (1) acts consistently in the public interest, and (2) takes steps to let his or her community know, through appropriate communications techniques and channels, that he functions in the community's interest. It could be more simply stated: Doing good, and getting credit for it. Public relations is different from advertising in that it usually imparts its message by indirection and, therefore, more subtly than advertising, and it normally is not paid for in the manner that advertising is. It confronts the reader, listener or viewer on a different credibility level than paid advertising and ideally is in the form of legitimate news or public interest information. Public relations, or "PR" as it is often abbreviated, has taken a bad rap in recent years as tending to mean something that is gimmicky or "staged" to make something or somebody "look" better than it really is. There are charlatans in the public relations craft just as there are quack doctors, but there also are many, many more highly responsible and highly professional public relations practitioners.

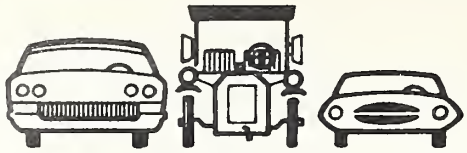
Market Research — The fact-finding process to determine what is the length, breadth, depth and nature of the market to be reached and served. It relates to the first part of the 2-part process called marketing. It is part of defining the problem. When market research mutates into market(ing) research, it refers not to the market or markets themselves, but to the nature of the communications approaches, methodologies or techniques to be used to reach and service the market(s). Techniques used in market(ing) research range from highly scientific statistical studies with strict control of variables to attitude and public opinion surveys that leave considerable room for interpretation. As with any other kind of research, it is far from infallible — the Edsel was launched in the wake of massive market studies. However, it is a sine qua non of any intelligent marketing/communications program.

Merchandising — Activities designed to call attention to and/or define comparatively the product or service you wish to make available to the market. This includes packaging, promoting, advertising and, yes, pricing — all in as coordinated and effective a fashion as possible. In the case of medical services this is an ongoing process but requires consistent attention in terms of competitive merchandising activities. Not only the quality and attractiveness of your personal services are involved here, but also the attractiveness and comfort of your office surroundings, courtesy and concern of your staff, efficiency of your office systems, functionality and efficiency of your equipment and instrumentation, etc. Merchandising is always putting your best foot forward.

Publicity — One of the primary components of an active, effective public relations program for your practice. We have defined public relations as partly attitudinal and partly functional, "Doing good, and getting credit for it." The first part relates to your attitude toward yourself, your profession and people generally; the second must be made to happen. Publicity is one of the basic ways to do this. Technically, publicity consists of various means of making known to others, i.e., the news media, things that you do, your practice does or your specialty does that are in the public interest. Obviously, this must be done with subtlety and good taste, but it can be done. For example, if you plan to have an open house, take in a new associate, or lead a community fund drive, these are legitimate reasons for making the activity known to the local news media. This "making known" can be handled in many different ways — the best known is the classic "press release." However, it may not be the most appropriate. Rather, a phone call with the facts would be more informal and acceptable. Much depends on the individual professional, the nature of the situation, a large number of variables. Suffice to say that an understanding and appreciation of what can be accomplished by open and ongoing communications, including publicity, is critical. Help is readily available in specific circumstances.

Audit — A review or appraisal of your practice situation, in terms of the basic marketing definition — determining your and your patients' needs, and how best to satisfy those needs. This means a careful and personal appraisal of personnel, procedures, systems, comparative fees and insurance practices, etc. It also means a review of your market area to determine possible demographic shifts, access changes such as bus line and schedule revisions or new road or shopping center construction, etc. These structural changes or population shifts could be going on under your nose without your realizing their potential impact on your practice. An audit also would bring to light alternative forms of health care delivery that could impact your practice. Such a practice appraisal is something the alert, marketing-oriented physician should consider on an ongoing basis, or at least periodically.

(Marketing Glossary Continued Next Month)



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South Dakota State Medical Association Annual Meeting

June 7-10, 1984

Howard Johnson Motor Lodge
Rapid City, South Dakota

Scientific Sessions
and
Risk Management

South Dakota Society Of Pathologists

Officers for 1983-84

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Child Automobile Safety Restraints

No parents would want to be accused of wantonly exposing their child to debilitating illnesses or to potentially lethal diseases. Yet many are doing just that by failing to protect their children in automobiles. It is a well known fact that the majority of automobile related injuries and fatalities occur to adults who are not wearing seatbelts, or to children without safety restraints. In 1982, four children under four years of age were killed in motor vehicle traffic accidents in South Dakota. The unknown factor is the number of children who were injured, suffering either minor or permanently disabling sequela. A ten pound baby becomes a 300 pound projectile when a car going 30 miles an hour comes to a sudden stop. This is the same impact that would occur from a fall from a three story building. A baby being held by an adult, who is not wearing a seat belt, would be crushed by a force of 3,600 pounds. In South Dakota, between 1976-1981, no child aged 0-5 years was killed when known to be wearing a safety restraint. According to the South Dakota Department of Public Safety, Division of Highway Safety, in 1981 85% of children under six years of age who were injured in motor vehicle accidents were not wearing safety restraints. Contrary to the myth that infants and young children are small and flexible and less apt to be injured than adults, they are more vulnerable to head injuries than adults. As a result, they have a higher risk of serious injury.

When considering morbidity and mortality strategies preventability must be given first consideration. The American Academy of Pediatrics and the South Dakota Pediatric Society are vigorous advocates of child restraint laws. At present, 41 states and the District of Columbia have mandated child safety seat usage. South Dakota is not one of these states. The effects of these safety laws have not been measured but reports from Canada indicate that the legal requirements of safety restraints does save lives.

A bill concerning child restraint, endorsed by the South Dakota State Medical Society, was introduced by Senator Robert Giebink in the South Dakota Legislature in 1983. Another bill, also endorsed by the Medical Society, has been introduced again this year.

Beginning in January, 1984, the South Dakota Department of Health and the South Dakota Department of Public Safety will co-sponsor a program to increase the usage of infant safety seats. The goal of this program is to reduce infant mortality and morbidity secondary to traffic accidents. An educational conference was held in Mitchell in January. A comprehensive list of local groups who are currently operating a safety seat program or are interested in developing such a program is being compiled. Statewide participation in such a program is encouraged. Local groups who participate in the Safety Seat Program will be provided two infant seats for each seat provided by the local organization. Also, educational programs will be provided to any local group upon request. Robert J. Graff, Health Services Assistant Administrator, South Dakota State Department of Health, 523 East Capitol, Pierre, SD 57501, may be contacted for further information.

Primary prevention is available for only a relatively few life threatening situations. We, as a profession, vigorously support preventing disability and death and should direct this same vigor in the support of the use of car safety seats.

Willis F. Stanage, M.D.
John B. Gregg, M.D.
Lawrence J. Massa

References

1. Wegman ME; Annual Summary or Vital Statistics, **Pediatrics.** 1983;76:6.
2. SDSDH, State Planning and Development Agency, **Time to Live.** Dec. 1982.
3. SDSDH, Center for Health Statistics. Vital Statistics and Health Status, 1982.

SD *Auxiliary News*

EVER WONDER WHERE YOUR MONEY IS GOING when you make a contribution to AMA-ERF? The AMA Auxiliary's AMA-ERF Committee sent a letter to medical school deans to find out just that. Over half of the deans responded, and a statistical report was compiled. The results show that:

—Twelve use the funds strictly for scholarships, loan programs, and aid for needy students.
—Nine split the money between student loans and student activities including emergency short-term loans, interviews, and publication printing.
—Two schools use the money entirely for the medical school library.
—Four divide funds between student activities and school programs covering tutoring and teaching aids for students, faculty retention, and curriculum evaluation.
—Student research programs account for another two schools which responded.

The American Medical Association Education and Research Foundation received more than $2 million in gifts by the AMA Interim Meeting, reported its president, Rufus K. Broadaway, MD, at the meeting in Los Angeles. The foundation received almost $80,000 in contributions to the new Medical Student Assistance Fund during the first three quarters of the year. Contributions to the student assistance fund may be designated for specific medical schools and are restricted to student financial aid programs at the schools.

Dr. Broadaway expressed gratification to the AMA Auxiliary for its "tireless efforts in behalf of the AMA-ERF."

Marie Hovland

Marie Hovland, President
South Dakota State Medical Auxiliary

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SD *Future Meetings*

March

Nuclear Cardiology, Vacation Village Hotel, San Diego, CA, Mar. 1-3. 24 hrs. Category I credits. Contact: Nat'l Instit. for Prof. Education, 4880 River Bend Rd., Boulder, CO 80301. Phone: 800-421-8452.

Seventh Annual Vail Cancer Treatment Conference, Lion Square Lodge, Vail, CO, Mar. 3-10. Fee: $335. 22 hrs. AAFP & AMA Category 1 credits. Contact: Vail Cancer Treatment Conf., P.O. Box 11338, Denver, CO 80211-0338. Phone: 800-525-5810.

Big Sky Lung Summit, Big Sky Resort, Big Sky, MT, Mar. 21-24. Fee: $100. Contact: Am. Lung Assoc. of Montana, 825 Helena Ave., Helena, MT 59601. Phone: (406) 442-6556.

April

Practical Cardiology for the Family Physician — 1984, Sheraton World, Orlando, FL, Apr. 2-4. 13.5 hrs. AMA Category I credits. Contact: Ronald J. Sanchez, Dir. of Comm., 9111 Old Georgetown Rd., Bethesda, MD 20814. Phone: (301) 897-5400.

Management and Theory of Pain in Cancer Patients, Four Seasons Hotel, Houston, TX, Apr. 5-7. Contact: Office of Conf. Services, Box 131, M. D. Anderson Hosp. & Tumor Instit., 6723 Bertner Ave., Houston, TX 77030. Phone: (713) 792-2222.

Cervical Neoplasia, Stouffers Hotel, Houston, TX, Apr. 6-7. Contact: Off. of Conf. Serv., Box 131, M. D. Anderson Hosp. & Tumor Instit., 6723 Bertner Ave., Houston, TX 77030. Phone: (713) 792-2222.

The Spectrum of Colon and Rectal Diseases in Primary Care, Hyatt Regency, Minneapolis, MN, Apr. 13-14. Fee: $170. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Iowa Dermatological Society — Spring Meeting, U. of Iowa Coll. of Med., Iowa City, IA, Apr. 13-14. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Tenth Annual Conference on Perinatal Medicine, Des Moines, IA, Apr. 17-18. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Radiation Therapy Seminar, U. of Iowa Coll. of Med., Iowa City, IA, Apr. 19. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Otolaryngology Clinical Conference, U. of Iowa Coll. of Med., Apr. 20. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Obstetrics-Gynecology Postgraduate Conference, U. of Iowa Coll. of Med., Apr. 23-24. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Family Practice Review: Update 1984, Holiday Inn Downtown, Minneapolis, MN, Apr. 23-27. Fee: $375. AMA Category I credits. Contact: CME, Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Health Life Cycles: Annual Women in Medicine Symposium, Malcolm Moos Hlth. Sci. Tower, U. of Minn., Minneapolis, MN, Apr. 27-28. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

May

Cardiology Today, U. of Iowa Coll. of Med., Iowa City, IA, May 1-4. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Cardiac Pacing, Sheraton-Midway, St. Paul, MN, May 4. AMA Category I Credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Endocrinology in Primary Care Clinical Practice, London, England, May 5-13. Contact: Cynthia Vaughn, Hlth. Sci. Seminars, P. O. Box 22023, San Francisco, CA 94122. Phone: (415) 861-2713.

Total Management of the Acute Myocardial Infarction, Battenfeld Aud., U. of Kan. Med. Ctr., May 7-8. Fee: $200. 13.25 hrs. AMA Category I credit & 12.5 hrs. AAFP credit. Contact: Jan Johnston, Off. of CME, U. of Kan. Med. Ctr., Rainbow & Olathe Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

Nuclear Magnetic Resonance 1984-National Symposium, Hyatt Regency Grand Cypress Resort, Orlando, FL, May 7-11. Fee: $350. 30 hrs. AMA Category 1 credit. Contact: Norine Karwel, Ed. Symposia, Box 17241, Tampa, FL 33682. Phone: (813) 879-8765.

Allergy and Clinical Immunology, Mayo Mem. Aud, Minneapolis, MN, May 10-12. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Iowa Cardiology, U. of Iowa Coll. of Med., Iowa City, IA, May 11. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

ENT in Primary Care: A Workshop, St. Joseph's Hosp., St. Paul, MN, May 11. 7 hrs. AMA Category I credits. Contact: Charles W. Drage, M.D., Dir. of CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

References:
1. Stone PH, Turi ZG, Muller JE. Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104:672-681, September 1982.
2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302:1269-1273, June 5, 1980.

BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug Interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related: Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request.

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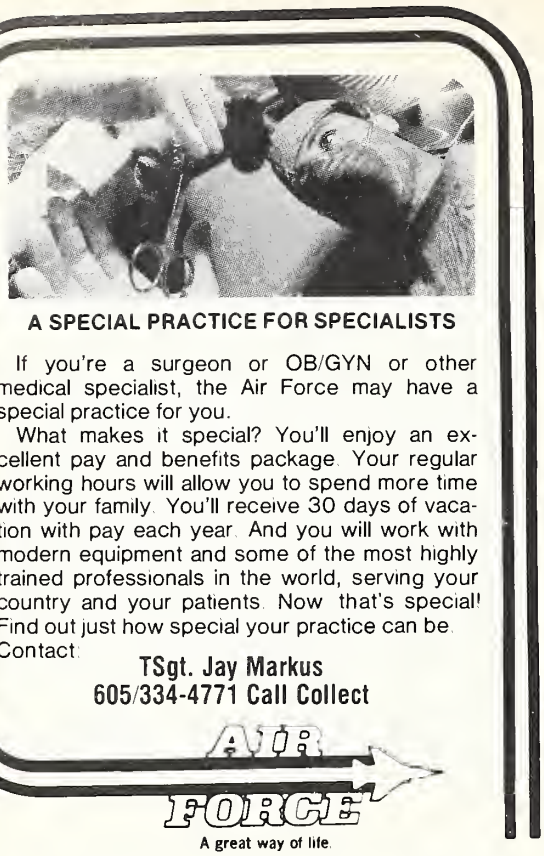

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PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again.

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).

for the varied faces of angina

PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

*Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page.*

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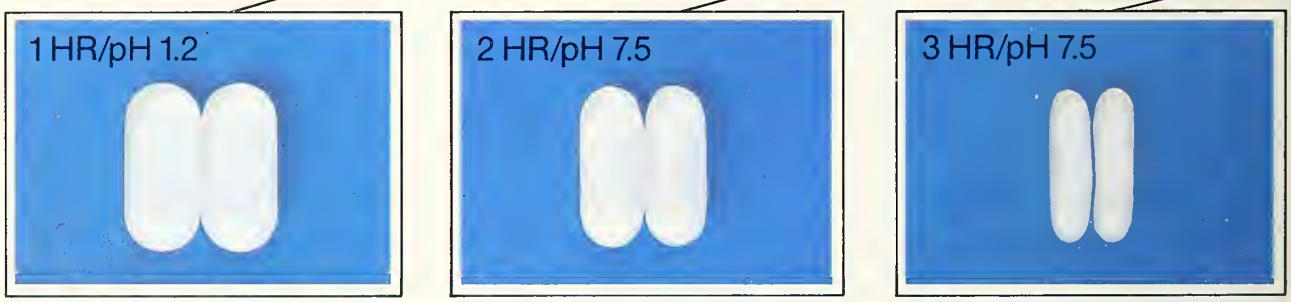

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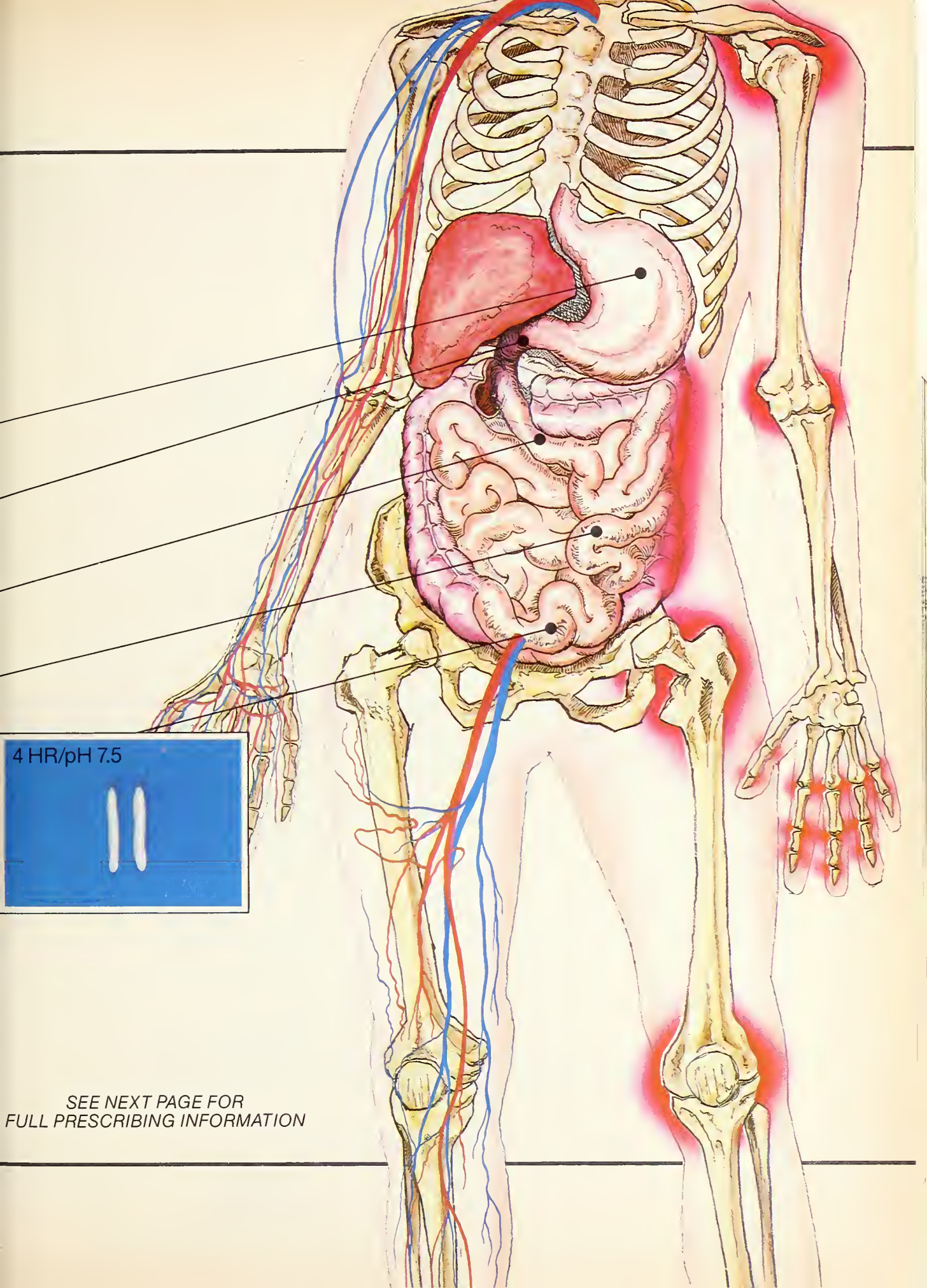
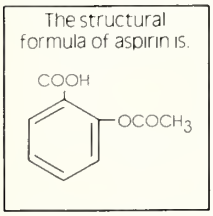


SEE NEXT PAGE FOR FULL PRESCRIBING INFORMATION

ZORprin (ASPIRIN) Zero-Order Release

DESCRIPTION: Each capsule-shaped tablet of Zorprin contains 800 mg of aspirin, formulated in a special matrix to control the release of aspirin after ingestion. The controlled availability of aspirin provided by Zorprin approximates zero-order release; the *in vitro* release of aspirin from the tablet matrix is linear and independent of the concentration of the drug. □ **CLINICAL PHARMACOLOGY:** Aspirin, as contained in Zorprin, is a salicylate that has demonstrated anti-inflammatory and analgesic activity. Its mode of action as an anti-inflammatory and analgesic agent may be due to the inhibition of synthesis of prostaglandins, although its exact mode of action is not known. □ Zorprin dissolution is pH-dependent. *In vitro* studies have shown very little aspirin to be released in acidic solutions, whereas, Zorprin releases the majority of its aspirin (90%) in a zero-order mode at a neutral to alkaline pH. It is this pH dependence of Zorprin that reduces direct contact between aspirin and the gastric mucosa, resulting in a reduction of its gastrointestinal side-effect potential. □ Bioavailability data for Zorprin have confirmed that plasma levels of salicylic acid and acetylsalicylic acid can be measured 24 hours after a single oral dose. This substantiates a twice daily dose regimen. Multiple dose bioavailability studies showed similar steady-state salicylate levels for Zorprin as for conventional release aspirin using the same total daily dose. Long-term monitoring of salicylate levels showed no signs of accumulation once steady-state levels were reached (4-6 days). □ Studies of *in vivo* prostaglandin levels (PGE2) have shown Zorprin plasma levels of salicylic acid and acetylsalicylic acid to reduce PGE2 levels 14 hours after a single oral 800 mg dose while an equivalent dose of aspirin produced a reduction of PGE2 levels only through six hours. Zorprin's effect on prostaglandins other than PGE2 has not been determined. □ Salicylates are excreted mainly by the kidney, and from studies in humans it appears that salicylate is excreted in the urine as free salicylic acid (10%); salicyluric acid (75%) salicylic phenolic (10%); acyl glucuronides (5%) and gentisic acid (<1%). □ **INDICATIONS & USAGE:** Zorprin is indicated for the treatment of rheumatoid arthritis and osteoarthritis. The safety and efficacy of Zorprin have not been established in those rheumatoid arthritic patients who are designated by the American Rheumatism Association as Functional Class IV (incapacitated, largely or wholly bedridden, or confined to wheelchair, little or no self-care). □ In patients treated with Zorprin for rheumatoid arthritis and osteoarthritis, the anti-inflammatory action of Zorprin has been shown by reduction in pain, morning stiffness and disease activity as assessed by both the investigators and patients. □ In clinical studies in patients with rheumatoid arthritis and osteoarthritis, Zorprin has been shown to be comparable to conventional release aspirin in controlling the aforementioned signs and symptoms of disease activity and to be associated with a statistically significant reduction in the milder gastrointestinal side effects (see ADVERSE REACTIONS). Zorprin may be well tolerated in some patients who have had gastrointestinal side effects with conventional release aspirin, but these patients when treated with Zorprin should be carefully followed for signs and symptoms of gastrointestinal bleeding and ulceration. □ Since there have been no controlled trials to demonstrate whether or not there is any beneficial effect or harmful interaction with the use of Zorprin in conjunction with other nonsteroidal anti-inflammatory agents (NSAI), the combination cannot be recommended (see **Drug Interactions**). □ **Because of its relatively long onset of action, Zorprin is not recommended for antipyresis or for short-term ahalgesia.** □ **CONTRAINDICATIONS:** Zorprin should not be used in patients known to be hypersensitive to salicylates or in individuals with the syndrome of nasal polyps, angioedema, bronchospastic reactivity to aspirin, renal or hepatic insufficiency, hypoprothrombinemia or other bleeding disorders. Zorprin is not recommended for children under 12 years of age; it is contraindicated in all children with fever accompanied by dehydration. □ **WARNINGS:** Zorprin should be used with caution when anticoagulants are prescribed concurrently, since aspirin may depress platelet aggregation and increase bleeding time. Large doses of salicylates may have hypoglycemic action and enhance the effect of the oral hypoglycemics, concomitant use therefore is not recommended. However, if such use is necessary, dosage of the hypoglycemic agent must be reduced. The hypoglycemic action of the salicylates may also necessitate adjustment of the insulin requirements of diabetics. □ While salicylates in large doses have a uricosuric effect, smaller amounts may reduce water excretion and increase serum uric acid. □ USE IN PREGNANCY: Aspirin can harm the fetus when administered to pregnant women. Aspirin interferes with maternal and infant hemostasis and may lengthen the duration of pregnancy and parturition. Aspirin has produced teratogenic effects and increases the incidence of stillbirths and neonatal deaths in animals. □ If this drug is used during pregnancy, or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus. □ Aspirin should not be taken during the last 3 months of pregnancy. □ **PRECAUTIONS:** Appropriate precautions should be taken in prescribing Zorprin for patients who are known to be sensitive to aspirin or salicylates. Particular care should be used when prescribing this medication for patients with erosive gastritis, peptic ulcer, mild diabetes or gout. As with all salicylate drugs, caution should be exercised in prescribing Zorprin for those patients with bleeding tendencies or those on anticoagulants. □ In order to avoid exacerbation of disease or adrenal insufficiency, patients who have been on prolonged corticosteroid therapy should have their therapy tapered slowly rather than discontinued abruptly when Zorprin is made a part of the treatment program. □ Patients receiving large doses of aspirin and/or prolonged therapy may develop mild salicylate intoxication (salicylism) that may be reversed by dosage reduction. □ Salicylates can produce changes in thyroid function tests. □ Salicylates should be used with caution in patients with severe hepatic damage, preexisting hypoprothrombinemia, Vitamin K deficiency and in those undergoing surgery. □ Since aspirin release from Zorprin is pH dependent, it may change in those conditions where the gastric pH has been increased as a result of antacids, gastric secretion inhibitors or surgical procedures. □ **Drug Interactions:** (See **WARNINGS**) Aspirin may interfere with some anticoagulant and antidiabetic drugs. Drugs which lower serum uric acid by increasing uric acid excretion (uricosurics) may be antagonized by the concomitant use of aspirin, particularly in doses less than 2.0 grams/day. Nonsteroidal anti-inflammatory drugs may be competitively displaced from their albumin binding sites by aspirin. This effect may negate the clinical efficacy of both drugs. Also, the gastrointestinal inflammatory potential of nonsteroidal anti-inflammatory drugs may be potentiated by aspirin. The combination of alcohol and aspirin may increase the risk of gastrointestinal bleeding. □ Aspirin may enhance the activity of methotrexate and increase its toxicity. □ Sodium excretion produced by spironolactone may be decreased in the presence of salicylates. Concomitant administration of other anti-inflammatory drugs may increase the risk of gastrointestinal ulceration. Urinary alkalinizers decrease aspirin's effectiveness by increasing the rate of salicylate renal excretion. Phenobarbital decreases aspirin's effectiveness by enzyme induction. □ **Pregnancy Category D.** See **WARNINGS** Section. □ **Nursing Mothers:** Salicylates have been detected in the breast milk of nursing mothers. Because of the potential for serious adverse reactions from aspirin in nursing infants, a decision should be made whether to discontinue nursing or discontinue the drug, taking into account the benefit of the drug to the mother. □ **ADVERSE REACTIONS: Hematologic:** Aspirin interferes with hemostasis. Patients with a history of blood coagulation defects or receiving anti-coagulant drugs or with severe anemia should avoid Zorprin. Aspirin used chronically may cause a persistent iron deficiency anemia. □ **Gastrointestial:** Aspirin may potentiate peptic ulcer, and cause stomach distress or heartburn. Aspirin can cause an increase in occult bleeding and in some patients massive gastrointestinal bleeding. However, the greatest release of active drug from Zorprin is designed to occur in the small intestine over a period of time. This has resulted in fewer symptomatic gastrointestinal side effects. □ **Allergic:** Allergic and anaphylactic reactions have been noted when hypersensitive individuals have taken aspirin. Fatal anaphylactic shock, while not common, has been reported. □ **Respiratory:** Aspirin intolerance, manifested by exacerbations of bronchospasm and rhinitis, may occur in patients with a history of nasal polyps, asthma, or rhinitis. The mechanism of this intolerance is unknown but may be the result of aspirin-induced shunting of prostaglandin synthesis to the lipoxygenase pathway and the liberation of leukotrienes, *e.g.* slow-reacting substance of anaphylaxis. □ **Dermatologic:** Hives, rashes, and angioedema may occur, especially in patients suffering from chronic urticaria. □ **Central Nervous System:** Taken in overdoses, aspirin provides stimulation which may be manifested by tinnitus. Following initial stimulation, depression of the central nervous system may be noted. □ **Renal:** Aspirin rarely may aggravate chronic kidney disease. □ **Hepatic:** High doses of aspirin have been reported to produce reversible hepatic dysfunction. □ **OVERDOSAGE:** Overdosage, if it occurs, would produce the usual symptoms of salicylism: tinnitus, vertigo, headache, confusion, drowsines, sweating, hyperventilation, vomiting or diarrhea. Plasma salicylate levels in adults may range from 50 to 80 mg/dl in the mildly intoxicated patient to 110 to 160* mg/dl in the severely intoxicated patient. An arterial blood pH of 7.1 may indicate serious poisoning. The clearance of salicylates in children is much slower than adults and should receive due consideration when aspirin overdosages occur in infants; salicylate half-lives of 30 hours have been reported in infants 4-8 months old. Treatment for mild intoxication should include emptying the stomach with an emitic, or gastric lavage with 5% sodium bicarbonate. Individuals suffering from severe intoxication should, in addition, have forced diuresis by intravenous infusions of sodium bicarbonate and dextrose or sodium lactate. In extreme cases, hemodialysis or peritoneal dialysis may be required. □ (*A plasma salicylate level of 160 mg/dl in an adult is usually considered lethal.) □ **DOSAGE & ADMINISTRATION:** *In order to achieve a zero-order release, the tablets of Zorprin should be swallowed intact.* □ *Breaking the tablets or disrupting the structure will alter the release profile of the drug.* □ *It is recommended that Zorprin be taken with sufficient quantities of fluids (8 oz. or more).* □ **Adult Dosage:** For mild to moderate pain associated with rheumatoid arthritis and osteoarthritis, the recommended initial dose of Zorprin is 1600 mg (2-800 mg tablets) twice a day. Because of Zorprin's prolonged release of aspirin into the bloodstream, Zorprin tablets may be taken as a b.i.d. dose. Further adjustment of the dosage should be determined by the physician, based upon the patient's response and needs. Since it will take 4-6 days to reach steady-state levels of salicylic acid with Zorprin, it is recommended dosages be given for at least one week before further adjustment. In general, patients with rheumatoid arthritis seem to require higher doses of Zorprin than do patients with osteoarthritis. □ **Zorprin is not recommended for children below the age of 12.** □ **HOW SUPPLIED: Zorprin Tablets 800 mg;** plain, white capsule-shaped tablets. □ Bottles of 100 Tablets —NDC 0524-0057-01. □ **Caution:** Federal law prohibits dispensing without prescription. □ U.S. Patent No. 4,308,251. □ **Manufactured and Distributed by: BOOTS PHARMACEUTICALS, INC., Shreveport, Louisiana 71106 U.S.A.**



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Volume XXXVII March 1984 Number 3

SOUTH DAKOTA JOURNAL OF Medicine

Treatment of Depression in Primary Care: Decision-Making Aided by an Algorithm

Clinicopathological Conference Fifty Year Old Caucasian Female With Abdominal Pain and Headache

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References:
1. Stone PH, Turi ZG, Muller JE: Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104:672-681, September 1982.
2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302:1269-1273, June 5, 1980.

BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

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Volume XXXVII/No. 3 March 1984 (ISSN 0038-3317)

Official Publication of the
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the South Dakota Chapter
of The American Academy
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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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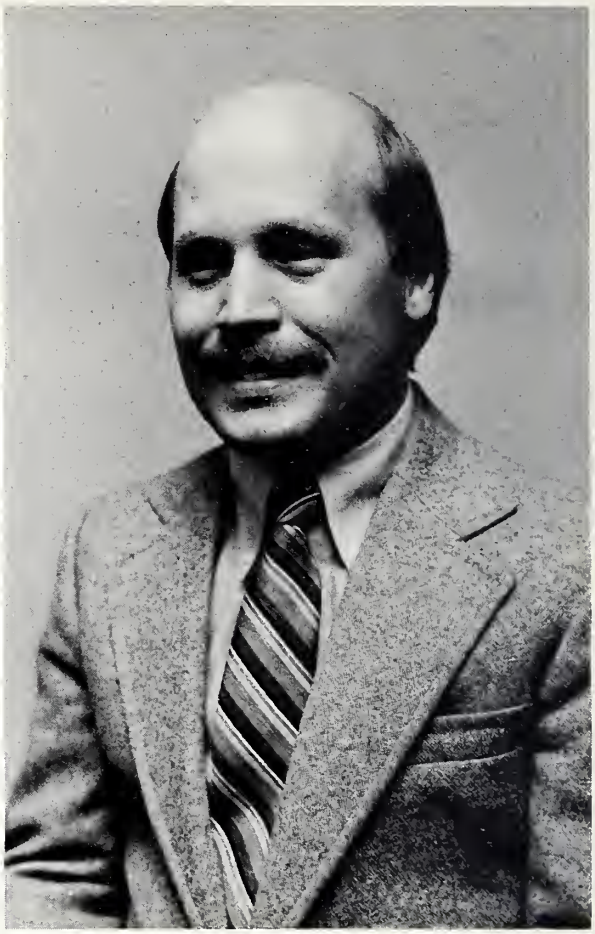

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Treatment of Depression in Primary Care: Decision-Making Aided by an Algorithm

Vinod Bhatara, M.D., M.S., F.R.C.P. (c)*

ABSTRACT

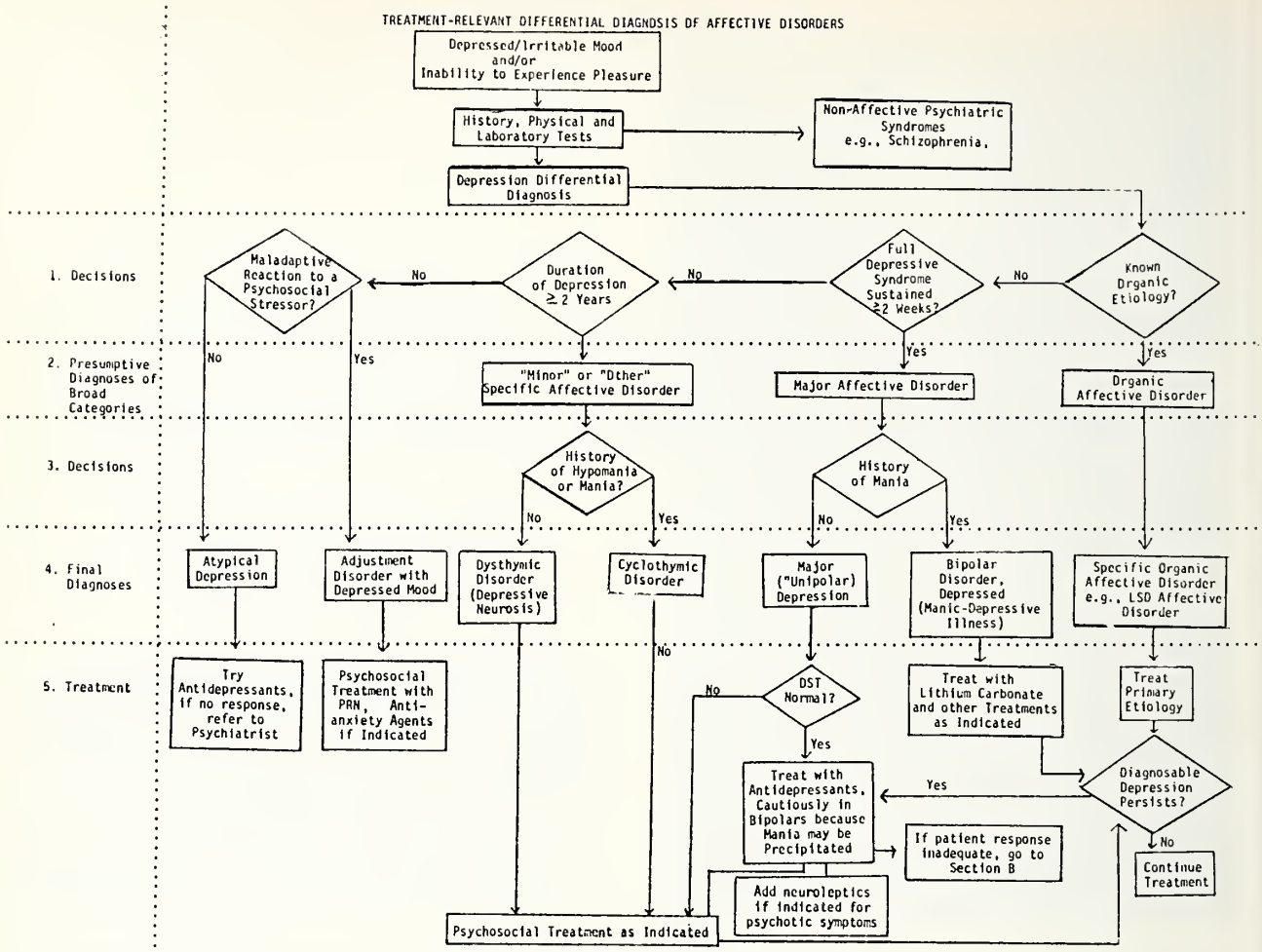
Diagnosis and treatment of depression in primary care are viewed as major components of a sequential decision-making process. Such a decision-making procedure can be clearly represented by an algorithm (decision-tree or flow chart). The focus of this paper is an algorithmic approach to clinical decision-making in depression. Using an algorithm, written to illustrate the diagnosis and treatment of depression in primary care, the article presents a rational approach in thinking about affective disorders. The algorithmic approach to clinical decision-making in depression is presented as a supplement to, and not as a replacement for, more familiar aids to clinical judgement. Management of some common side effects of antidepressants is also displayed.

Several studies have shown that about 55 percent of persons with mental health problems are treated by primary care physicians.[1, 2] It has also been found that in primary care, pharmacological therapy is the treatment modality most often used to treat mental disorders. Therefore, it is crucial that the training needs of primary care physicians in pharmacologic treatment of a common disorder such as depression are met. Unfortunately, textbooks fail to fulfill these training needs. As Margolis notes, "It seems that a text describes one reality in which the physician is a passive recipient of knowledge about diseases, while the clinician is required to function in a different reality, one that consists of making sequential decisions. In contrast to the textbook, the clinical algorithm, or flow chart, is a graphic format that sets forth a stepwise procedure for making decisions about the diagnosis and treatment of a clinical problem."[3] Essex and Gosling have even developed an algorithmic method for management of mental health problems in cross-cultural settings. The evaluation studies that have been done in cross-cultural settings "confirm that the method is acceptable and efficient, and gives management decisions with a high degree of repeatability."[4]

Like treatment of any other syndrome, the treatment of depression in primary care involves a sequence of diagnostic and therapeutic decisions. Such a sequential clinical decision-making process is most clearly represented by means of an algorithm (flow chart of a decision-tree). Using an algorithm written to illustrate diagnosis and treatment of depression in primary care, the article presents a rational approach in thinking about affective disorders. The algorithmic approach to the diagnosis and treatment of depression in medical practice is presented as a supplement to, and not as replacement for, more familiar aids to clinical judgement.

A review of literature revealed several algorithms on depression, but none that used the latest diagnostic terminology and also integrated diagnosis and treatment at the same time.[5-8] Only the flow chart in the third edition of Diagnostic and Statistical Manual (DSM III) used the latest diagnostic terms in depression. This paper presents an algorithm that 1) makes use of the latest advances in diagnosis, 2) makes explicit relationship between diagnosis and treatment, and 3) is geared to the primary care level.

* Associate Professor and Director of Research, USD School of Medicine, Department of Psychiatry, 2501 W. 22nd St., Sioux Falls, SD 57105.


TREATMENT-RELEVANT DIFFERENTIAL DIAGNOSIS OF AFFECTIVE DISORDERS
Depressed/Irritable Mood and/or Inability to Experience Pleasure
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Non-Affective Psychiatric Syndromes e.g., Schizophrenia,
Depression Differential Diagnosis
1. Decisions
2. Presumptive Diagnoses of Broad Categories
3. Decisions
4. Final Diagnoses
5. Treatment
Maladaptive Reaction to a Psychosocial Stressor?
Duration of Depression ≥ 2 Years
Full Depressive Syndrome Sustained ≥2 Weeks?
Known Organic Etiology?
No
Yes
"Minor" or "Other" Specific Affective Disorder
Major Affective Disorder
Organic Affective Disorder
History of Hypomania or Mania?
History of Mania
Atypical Depression
Adjustment Disorder with Depressed Mood
Dysthymic Disorder (Depressive Neurosis)
Cyclothymic Disorder
Major ("Unipolar") Depression
Bipolar Disorder, Depressed (Manic-Depressive Illness)
Specific Organic Affective Disorder e.g., LSD Affective Disorder
Try Antidepressants, if no response, refer to Psychiatrist
Psychosocial Treatment with PRN, Anti-anxiety Agents if Indicated
DST Normal?
Treat with Lithium Carbonate and other Treatments as Indicated
Treat Primary Etiology
Treat with Antidepressants, Cautiously in Bipolars because Mania may be Precipitated
Diagnosable Depression Persists?
If patient response inadequate, go to Section B
Continue Treatment
Add neuroleptics if indicated for psychotic symptoms
Psychosocial Treatment as Indicated

Figure 1, Section A

An algorithm is presented in Figure 1 (A and B) to illustrate the flow of diagnostic and therapeutic decision-making in affective disorders. To keep the algorithm simple and readable, the treatment of such depressions as atypical depressions that are infrequently treated by primary care physicians is not described. Management of some common side effects of antidepressants is displayed in Figure 2. Because of the space limitations (and to keep the decision-tree manageable), such rare effects of antidepressants as agranulocytosis are omitted. The physician should, of course, be aware of the rarer side effects of antidepressants and implement appropriate counter measures whenever necessary.

In Figure 1, decision points are represented by diamonds. Section A of Figure 1 displays treatment-relevant differential diagnosis of affective disorders. In many cases of depression,[5] the presenting complaint is not depressed mood, but irritability or anhedonia (inability to experience pleasure). Thus DSM III acknowledges the existence of "masked depressions." The major focus of initial clinical examination is to rule out such non-affective psychiatric syndromes as schizophrenia, which are often associated with depressed mood, and to exclude etiologically-relevant organicity (disease or drug). In cases when depressions are associated with or "secondary to" a physical disease, substance use, or a non-affective disorder, the treatment of choice is the treatment of the primary etiology. For instance, the depression associated with hypothyroidism should be treated with thyroid-replacement therapy. However, if diagnosable depression persists in spite of adequate treatment of hypothyroidism, treatment with antidepressants may be indicated, provided there is no contraindication to the use of antidepressants.

If the criteria for a full depressive syndrome as described in DSM III[5] — Table I are met, then the presumptive diagnosis of a major affective disorder can be made.

Major affective disorders can be subdivided into unipolar and bipolar affective disorders on the basis of history of mania in bipolar affective disorder (previously called manic-depressive illness). The treatment of choice in bipolar affective disorder (for prophylaxis of recurrence and for acute manic episodes, but not for acute depression) is lithium car-

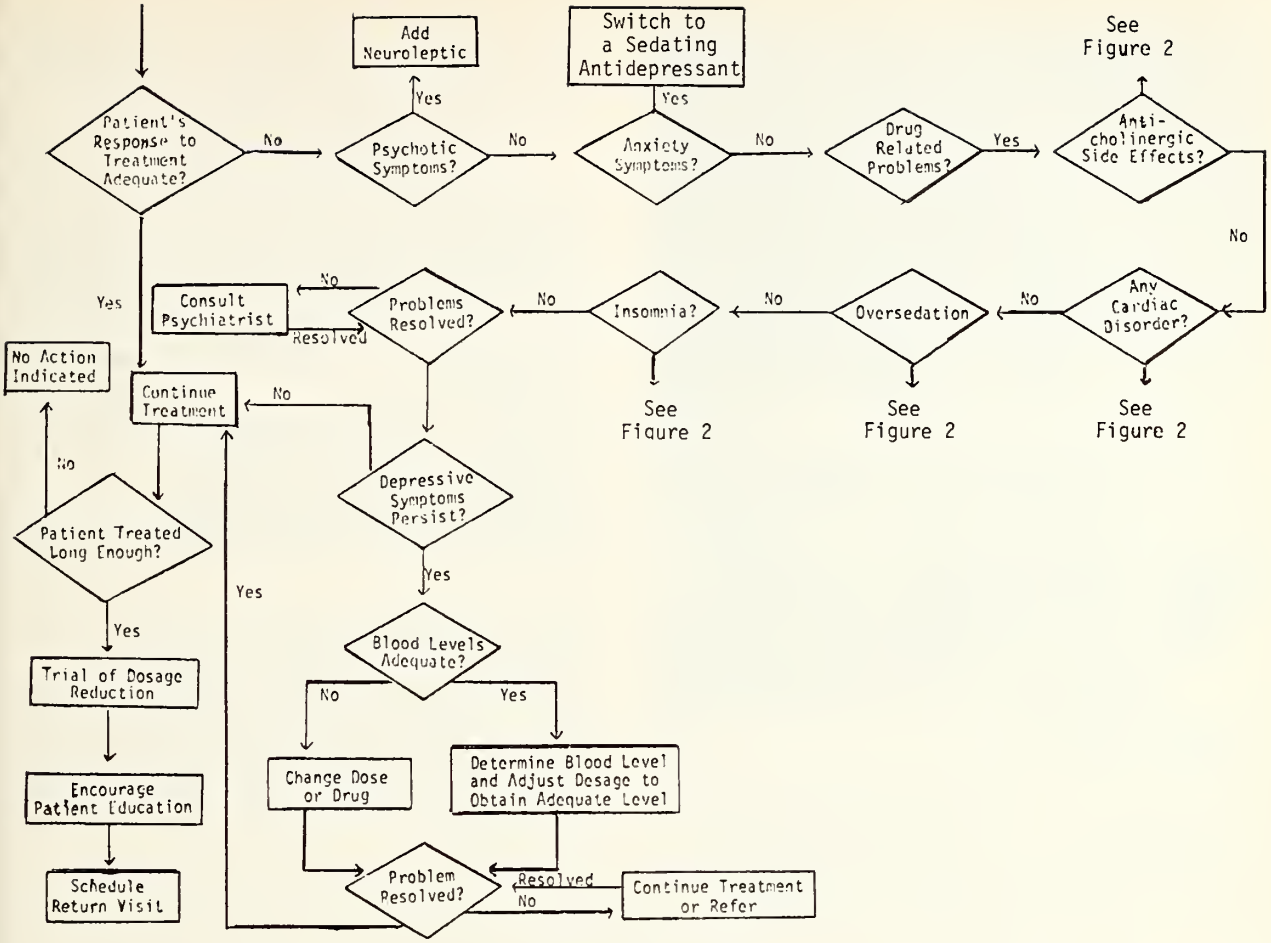

Figure 1, Section B

bonate. Effectiveness of lithium carbonate in treatment of acute depressive eipsodes of bipolar affective disorders has not been clearly established. In general, bipolar patients are difficult to manage in primary care, and should be referred to a phychiatrist for more specialized treatment. Depressed patients without history of mania (''unipolar'' major depressions), however, are more easily manageable with antidepressants. If such psychotic symptoms as hallucinations or delusions are associated with a major depression, then neuroleptics (antipsychotics) can be added.

Identification of patients who are likely to respond to antidepressants is based on 1) presence of a cluster of signs and symptoms — so called ''endogenous features'' as listed in Table I; and 2) diagnostic laboratory tests. The Dexamethasone Suppression Test (DST in Figure 1) has been found useful as a confirmationatory test for diagnosis of major depression and as an adjunct to assess and confirm response to antidepressant treatment. As Gold, et al state, ''While Dexamethasone Suppression Test may identify only half of the patients suspected clinically of having a unipolar depression, false positive findings are exceedingly rare.''[7] In their view, a positive DST, i.e., lack of cortisol suppression after Dexamethasone administration ''has considerable power and can guide the treatment.''

If the required number of criteria from DSM III for major affective disorder are not met, but only some of the criteria are met, a presumptive diagnosis of broad category ''minor'' or ''other'' Specific Affective Disorders can be made. Specific Affective Disorders category can be subdivided into Dysthymic and Cyclothymic disorders on the basis of history of mania or hypomanic in cylothymic disorder. The treatment of choice in these Specific Affective Disorders is psychosocial. However, antidepressants are indicated if depression does not respond to psychosocial measures alone. When depressed mood is chronologically associated with an identifiable stressor (e.g., death of a spouse), treatment is directed at disabling depression and anxiety, after the diagnosis of adjustment disorder with depressed mood (previously called situational depression) is made. The term adjustment disorder implies that the course of depression is determined by the stressor. If the stressor is a discrete event such as

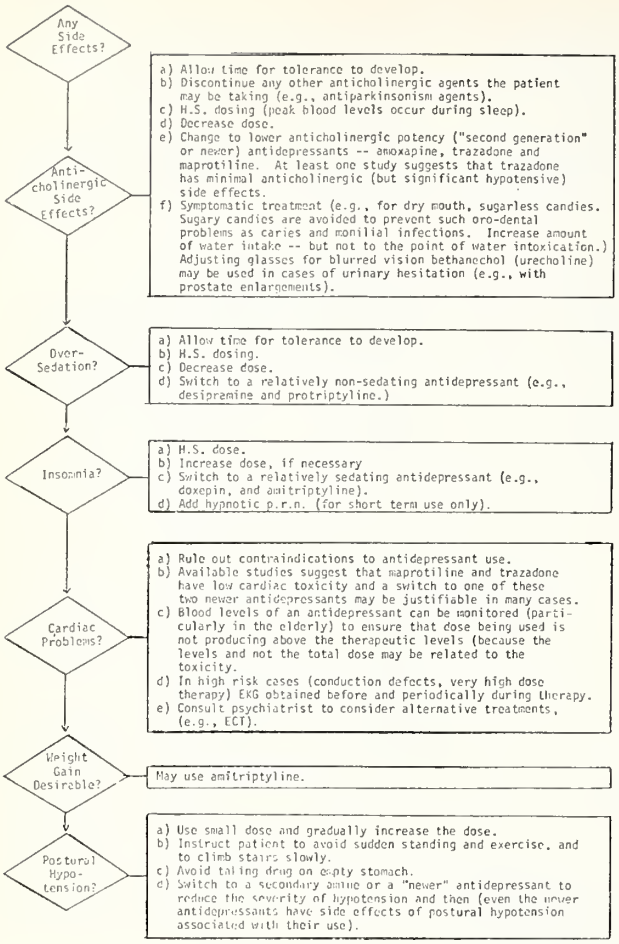
Any Side Effects?
a) Allow time for tolerance to develop.
b) Discontinue any other anticholinergic agents the patient may be taking (e.g., antiparkinsonism agents).
c) H.S. dosing (peak blood levels occur during sleep).
d) Decrease dose.
e) Change to lower anticholinergic potency ("second generation" or newer) antidepressants -- amoxapine, trazadone and maprotiline. At least one study suggests that trazadone has minimal anticholinergic (but significant hypotensive) side effects.
f) Symptomatic treatment (e.g., for dry mouth, sugarless candies. Sugary candies are avoided to prevent such oro-dental problems as caries and monilial infections. Increase amount of water intake -- but not to the point of water intoxication.) Adjusting glasses for blurred vision bethanechol (urecholine) may be used in cases of urinary hesitation (e.g., with prostate enlargements).
Anti-cholinergic Side Effects?
Over-Sedation?
a) Allow time for tolerance to develop.
b) H.S. dosing.
c) Decrease dose.
d) Switch to a relatively non-sedating antidepressant (e.g., desipremine and protriptyline.)
Insomnia?
a) H.S. dose.
b) Increase dose, if necessary
c) Switch to a relatively sedating antidepressant (e.g., doxepin, and amitriptyline).
d) Add hypnotic p.r.n. (for short term use only).
Cardiac Problems?
a) Rule out contraindications to antidepressant use.
b) Available studies suggest that maprotiline and trazadone have low cardiac toxicity and a switch to one of these two newer antidepressants may be justifiable in many cases.
c) Blood levels of an antidepressant can be monitored (particularly in the elderly) to ensure that dose being used is not producing above the therapeutic levels (because the levels and not the total dose may be related to the toxicity.
d) In high risk cases (conduction defects, very high dose therapy) EKG obtained before and periodically during therapy.
e) Consult psychiatrist to consider alternative treatments, (e.g., ECT).
Weight Gain Desirable?
May use amitriptyline.
Postural Hypo-tension?
a) Use small dose and gradually increase the dose.
b) Instruct patient to avoid sudden standing and exercise, and to climb stairs slowly.
c) Avoid taking drug on empty stomach.
d) Switch to a secondary amine or a "newer" antidepressant to reduce the severity of hypotension and then (even the newer antidepressants have side effects of postural hypotension associated with their use).

Figure 2
Managing side effects.

being fired from a job, the duration of depression is likely to be brief — no more than a few months. Antidepressants are usually not indicated in adjustment disorder. Antianxiety[10] agents can be used for disabling dysphoria on as needed basis. Atypical depressions, which are refractory to antidepressants, perhaps are best referred to a psychiatrist. Monoamine oxidase[11] (MAO) inhibitor drugs are often indicated in atypical depressions.

Once the patient has been started on a specific antidepressant, the physician needs to find out whether or not the patient is responding to antidepressants adequately (Figure 1, Section B). Unresolved target symptoms can be resolved by the addition of another drug (e.g., a neuroleptic for psychotic symptoms) or by switching to another antidepressant (e.g., sedating antidepressant for unresolved anxiety symptoms). Management of specific drug side effects is displayed in Figure 2. However, if patient appears to be refractory to antidepressants, as shown by nonresponse or underresponse to an adequate trial (adequate dose for four weeks), antidepressant plasma levels should be measured. The levels should be adjusted to bring them into purported range of therapeutic concentration. If the patient's depression remains underresponsive, an antidepressant from a different class may be tried. For instance, if patient is nonresponsive to tricyclics, Trazadone can be administered.

TABLE I

*** Table I lists the signs and symptoms of the antidepressant responsive major depressions.**

†‡ Clinical Features of Antidepressant — Responsive Depression

(So called "Endogenous" Depression)

1) **Sleep disturbance is early morning awakening — at least two hours before the usual time.**
2) **Interest lost in nearly all activities because of inability to obtain pleasure from nearly all of these interests.**
3) **Guilt is excessive, inappropriate and may be delusional.**
4) **Reactivity to external events is so decreased that nothing pleases.**
5) **Constipation.**
6) **Anorexia — significant weight loss (1 lb/wk or 10 lb/year).**
7) **Psychomotor agitation or retardation.**
8) **Quality of depression distinct from grief.**
9) **AM depression (worse in the morning)**

* Listed in DSM III as symptoms of melancholia. Dexamethasone Suppression Test (DST) is also used to differentiate endogenous from exogenous depressions.

† Rund and Hutzler have created a mnemonic aid for remembering the above criteria: In Sad Cages. The letters stand for Interest (IN), Sleep Disorder (S), Appetite (A), Dysphoric Mood (D), Concentration Problems (C), Affect (A), Guilt (G), Energy (E), and SuicidaI Ideation (S). (Reference Rund and Hutzler: In Sad Cages: A Mnemonic for Depression. A letter to editor. **Am J Psychiatry,** 140:5, 1983.)

‡ Arranged in form of a mnemonic: SIG. R CAPS Q AN.

CONCLUSION

The article illustrates the application of algorithm to diagnostic and therapeutic decision-making in affective disorders. The decision-tree approach in thinking about depression can be further extended to the use of specific antidepressants. An algorithm can be written to raise and answer such questions as 1) How a specific antidepressant is to be selected; 2) What dosage of drug is to be used and for what duration; and 3) If maintenance treatment is indicated.

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President's Page

Common and sense often do not go together.
What makes sense is often not common.
What is common often does not make sense.
Together or apart, each require caution.
Lonna Guthmiller, R. N., 1982

Recently theft of laboratory animals from research centers on the east and west coasts has been reported in the press. One incident involved twelve dogs, of which five had surgically implanted pace makers worth up to $5,000 each. Six rats were stolen from the second center. While the loss has been estimated at $100,000, the cost in time and effort is more serious. Officials have denied the allegation that the animals were treated cruelly, and stated that care given them exceeded the requirements of current law regulating the use of animals in research. It is certain that scientists must maintain healthy animals and treat them humanely to obtain reliable research results. A group known as the Animal Liberation Front (ALF) claimed responsibility for both incidents.

In December and January severe weather in much of the nation caused starvation and suffering of animals, both wild and domestic. News media gave wide coverage to the problem, and reported widespread public response to the situation. The ALF was not singled out for outstanding action to help the animals.

A cardiologist at one of the centers described the action of the AFL as comparable to those of the Irish Republic Army, the Palestine Liberation Organization and other terrorists, "with the only difference being that human lives are not taken directly." Terrorism is always senseless, and pathetically common.

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S D *Clinicopathologic Conference*

Fifty Year Old Caucasian Female With Abdominal Pain and Headache

Kirk M. Odden, M.D.*
Discusser

John F. Barlow, M.D.†
Editor

Case # 996 558

This 50 year old married Caucasian female entered Sioux Valley Hospital with a chief complaint of abdominal pain and headache.

The patient had noted abdominal discomfort described as indigestion for some years which had been relieved by antacids. Three weeks prior to admission the patient noted more severe abdominal discomfort and one week prior to admission this was accompanied by epigastric discomfort and right lower quadrant tenderness and discomfort. There was mild nausea but no vomiting. The patient had anorexia and a small amount of weight loss. There had been no change in bowel habits but the stools had definitely become darker but without bright red blood. There was no diarrhea or constipation. During the previous week, the patient had developed fever and chills but the temperature had not been above 100°F. She did wake up at night wringing wet and had had sweats during the day as well. During the three days prior to admission she complained of continuous aching in the top of her head, not relieved by aspirin. There were no visual or other neurologic symptoms. There was also a five day history of pain in the upper lumbar area that radiated into both flanks but did not radiate into the hips or legs.

On the morning of admission the patient was awakened by severe right lower quadrant pain which caused her to sit up suddenly in bed. This gradually resolved. She was seen at the Family Practice Center and physical examination revealed epigastric tenderness. Hemoglobin was 12.7 gm/dl, white count 23,200/mm^3 (23.2 $\times$ 10^9/L) with 92% segmented neutrophils on differential, temperature 98.6°F, blood pressure 122 systolic and 82 diastolic, pulse 120/min and regular, weight 132 pounds. On rectal examination test was positive on stool for occult blood.

PAST MEDICAL HISTORY: The patient had had epigastric discomfort with aspirin products but could tolerate buffered aspirin. There was a history of allergy to sulfa, excision of benign breast cysts five years prior to admission, tubal ligation, and removal of a thyroid nodule many years prior to admission.

PHYSICAL EXAMINATION: The patient was upset and nauseated with headache and abdominal discomfort. Pulse 120/min and regular, respirations 14/min and regular, temperature 99.4°F, blood pressure 120 systolic and 84 diastolic. Examination of the head and neck was unremarkable. The chest was clear to auscultation and percussion. The heart was within normal limits of size with no murmurs or abnormal sounds. The peripheral pulses were normal. The breast examination was negative. There was diffuse discomfort in the lumbar and costovertebral angle regions on percussion without localization. The patient had pain and guarding in the epigastrium and left upper quadrant. The bowel sounds were normal and there were no palpable masses or organs in the abdomen. Neurologic examination was within normal limits. Rectal examination was negative except for a positive test on the stool for occult blood.

LABORATORY DATA: Urinalysis — light yellow, clear, specific gravity 1.004, pH 6.5, negative for protein, glucose, ketones, bilirubin, hemoglobin; sediment — negative. Hemoglobin — 12.4 gm/dl; hematocrit 38 vol/dl with normal red cell indices; white count 16,400/mm^3 (16.4 $\times$ 10^9/L) with 87% segmented neutrophils, 1% neutrophilic bands and 12% normal lymphocytes. Platelets — 508,000/mm^3 (518 $\times$ 10^9/L). The red cells appeared normochromic, normocytic on smear. A zetacrit was 75% (markedly elevated). Serum sodium was 138 meq/L, potassium 3.8 meq/L. Lactic dehydrogenase (LD), alkaline phosphatase, asparatate aminotransferase (SGOT, AST), total bilirubin, total protein, calcium, inorganic phosphorus, glucose, blood urea nitrogen, creatinine and uric acid levels were within normal limits in a 12-panel chemistry on serum. A total thyroxine was within normal limits. An electrocardiogram was within normal limits. A urine culture and three blood cultures showed no growth. A chest film was within normal limits. An upper gastrointestinal series showed a prominent filling defect in

* Resident in Family and Community Medicine, Sioux Falls Program Affiliated with USD School of Medicine, Sioux Falls, SD.
† Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

the distal antrum 5.2 × 3.8 cm (Fig. 1). An upper gastrointestinal endoscopy showed a probable extrinsic mass pressing on the posterior wall of the greater curvature of the stomach at least 5 cm in diameter. An operation was performed.

Figure 1
Filling defect in distal stomach (see protocol).

DR. ODDEN: The salient features of this case are:

1. History of abdominal discomfort, nausea, anorexia, chills and sweats.
2. Epigastric tenderness, mild guarding for positive tests for occult blood in the stool, leukocytosis and elevated zetacrit.
3. X-ray findings of a distal antral filling defect 5.2 × 3.8 cm.
4. Endoscopy showing a probable extrinsic mass pressing upon the posterior wall of the greater curvature of the stomach.

The history, physical examination and laboratory results are non-specific. The upper gastrointestinal x-ray series and gastroscopy are useful in that the circumscribed nature, large size and apparently intact mucosa or smooth surface over the mass all point toward an intramural benign stomach tumor or extrinsic mass. The following discussion focuses on stomach tumors.

In all series of gastric tumors, carcinoma is by far the most common tumor and malignant tumors outnumbered benign. A combined series had 87.9% carcinomas, 1.7% leiomyosarcomas, and 3% lymphomas. The other 7% consisted of benign tumors. The well circumscribed nature of this lesion would suggest one of the tumors in the benign category. Below is a table of some of the more common benign conditions which produce a filling defect (Table I).

Many benign tumors are small, 50% are less than 2 cm in diameter, and easily missed. Fifty percent of benign tumors are asymptomatic. Clinical manifestations depend upon size and location of the lesion and whether overlying mucosal ulceration has occurred. Ulcerated tumors may cause symptoms and signs similar to peptic ulcer. Most occur in the antrum and many develop at the pylorus so obstruction can occur and lead to further symptoms. Since symptoms, signs and x-ray findings are nonspecific, final diagnosis must be based on histologic characterization of each lesion. The x-ray findings and results of gastroscopy in this case help rule out mucosal abnormalities as seen in adenocarcinoma and adenomatous polyps. This still leaves us with a sizable list of rare conditions to discuss.

Statistics would indicate that a leiomyoma would be the most likely. The incidence in males and females is similar with an age range of 9-94 years. The site is the antrum in 39%, pylorus 12%, cardia 10% and fundus 12% of cases. It is a submucosal tumor with intact mucosa in half of the cases and an ulcerated surface in the other half. Bleeding is common as is epigastric pain. X-rays usually show an intramural filling defect with or without ulceration. Gastroscopy shows normal mucosa with or without ulceration. Leiomyomas cannot usually be differentiated from leiomyosarcoma although the former generally are small until resection and histologic examination has been performed since biopsy can be misleading.

Leiomyosarcomas constitute 1-2% of all gastric tumors and 10-30% of gastric sarcomas. There is a slight male predominence with an age range of 10-73 years. Symptoms and signs include epigastric pain, gastrointestinal bleeding, mass and obstruction. Most are intramural. Larger tumors may be up to 20 cm in diameter and may seem to be extragastric. Fifty percent of cases eventually fungate and ulcerate. Fifty percent have metastases beyond the stomach to liver, omentum, and the retroperitoneum.

Table I
BENIGN TUMORS OF THE STOMACH

	% All Gastric Tumors	% Incidence of Benign Tumors	% in Antrum	% >2 cm.	Maximum Diameter in Circum.
Adenomatous Polyp	3.1	44.7	51	85	11
Leiomyoma	2.0	29.4	39	9	40
Heterotopic pancreas	0.4	6.4	86	75	5
Vascular tumor	0.2	3.3	49	16	20
Neurogenic tumor	0.16	2.2	27	5	32
Lipoma	0.14	2.0	69	25	20

Direct extension is also common.

In terms of biologic behavior, leiomyoblastomas are an intermediate tumor between leiomyoma and leiomyosarcoma. They have a slight male predominence and bleeding is common. They are round or oval in shape and variable in size. Large tumors often ulcerate and may have areas of hemorrhage, necrosis and degeneration.

Lymphoma is the most common sarcoma of the stomach. It may present as an ulcerated or polypoid mass or as a diffuse lesion. Lymphoma is very unlikely in light of the x-ray and endoscopic findings.

Two developmental lesions must be considered — gastric duplication cyst and heterotopic (aberrant) pancreas. Duplication cysts can vary from cystic to tubular masses with or without communication with the digestive tract. In a series of 39 cases, 36 presented as spherical cystic masses. The lesions ranged from 1.3-10 cm and were located primarily along the greater curvature or on the distal third of the stomach producing compression of the gastric wall. Six of the 39 cases showed communication with the lumen of the stomach.

Heterotopic pancreas may be found in the gastric wall. About 60% occur in the stomach or duodenum and 85% of those located in the stomach are at the pylorus or in the antrum. They are most often submucosal and less frequently in the muscular wall. The size ranges from 1-5 cm with an intact surface of overlying gastric mucosa except for a small orifice seen on x-ray or a central depression or pit. They are usually asymptomatic but occasionally are associated with epigastric pain, nausea, vomiting, obstruction or bleeding.

Eosinophilic granuloma is an example of an inflammatory condition presenting as a gastric mass. They are polypoid, submucosal and usually found in the antrum. They are usually asymptomatic but may cause bleeding or obstruction. X-rays show an intramural mass lesion or an intraluminal pedunculated polyp that may be up to 9 cm in greatest dimension. A second condition, eosinophilic gastritis may present with diffuse infiltration of the gastric wall with eosinophils. Pain, vomiting and diarrhea are common. A history of allergy or asthma is often found and peripheral eosinophilia is common.

Vascular tumors are rare. The glomus tumor is the most frequent with an equal incidence in males and females. It is submucosal, found in the distal portion of the stomach and ranges in size from 1-4 cm. Bleeding secondary to ulceration is the most common symptom. Other vascular tumors include hemangiomas, lymphangiomas, and hemangiopericytomas.

The lipoma of the stomach is submucosal and located distally. The age range is 29-83 years. Ulceration of overlying mucosa leads to bleeding in 53%. Symptoms of obstruction occur in 33%. The average diameter is 5 cm.

Fibromas occur in the antrum in a submucosal location. The range in size is 3-8 cm.

Carcinoid tumors occur more commonly in the small bowel than the stomach. They usually have the highest incidence in the 6th decade, have intact mucosa, are 2-6 cm in size and 30% have metastases by the time of discovery.

Other tumors include neurofibromas, plasmacytomas, pseudolymphomas as well as the various forms of polyps. Extrinsic compression from pancreatic pseudocysts, accessory spleens, lesser sac abscesses, etc. could also account for the x-ray findings in this case.

In conclusion, this case most likely represents a tumor or tumor-like condition arising in the submucosa or intramural portion of the stomach. Adenocarcinoma, hyperplastic and adenomatous polyps and lymphoma are unlikely because of x-ray and gastroscopic findings. Leiomyoma and leiomyosarcoma are most likely statistically. However, one must consider other conditions as described earlier including duplication cysts and heterotopic pancreas. The final diagnosis, as in most cases of stomach masses, must be made on the basis of histologic findings.

Dr. Odden's Diagnosis
Benign Tumor of Stomach

DR. BARLOW: We received a segment of stomach measuring 8 cm along the lesser curvature and 17 cm along the greater curvature. On opening the specimen, on the anterior wall, 2 cm from the distal margin, was a firm circumscribed 4.5 x 3.5 x 3.0 cm mass (Fig. 2) which projected into the lumen and had an ulcerated surface. The tumor did not extend

Figure 2
Cross section of submucosal mass protruding into lumen of stomach with ulcerated surface.

beyond the wall of the stomach. Cut section revealed a homogeneous firm yellow surface. The tumor microscopically was well demarcated from the surrounding mucosa and muscularis. The tumor was quite vascular and was composed of a mixture of two types of cells — eosinophils and elongated cells consistent with fibroblasts or smooth muscle cells (Fig. 3). A Masson stain for connective tissue showed that the cells were indeed producing collagen suggesting that this was a fibroblastic lesion. At first, this tumor was thought to be a bizarre leiomyoma, but on further consideration, this represents the so-called fibrous inflammatory polyp of the gastrointestinal tract.

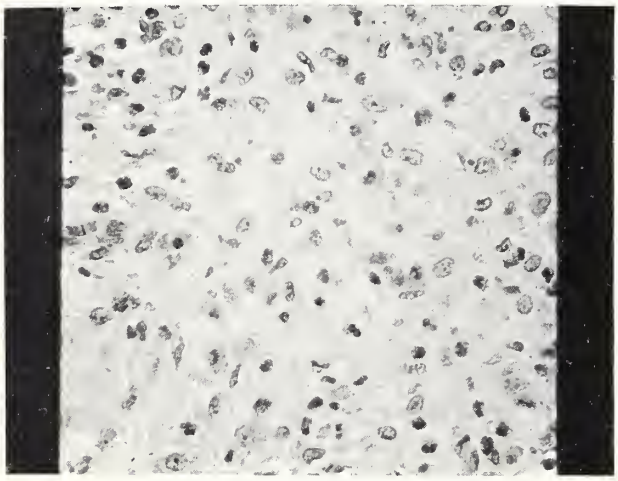

Figure 3
Microscopic view showing eosinophils and spindle cells characteristic of fibrous inflammatory polyp.

FINAL ANATOMICAL DIAGNOSIS FIBROUS INFLAMMATORY POLYP OF STOMACH

This lesion was first described by Vanek in 1949 as submucosal granuloma with eosinophilic infiltration. Johnstone and Morson reviewed a large series of 90 cases. The age range varied from 2-90 years with a mean of 53 years. There were 51 men and 38 women. Sixty of the lesions occurred in the stomach, most commonly in the antrum. Seventeen lesions appeared in the ileum, 5 in the colon, 4 in the jejunum, 3 in the duodenum and 1 in the esophagus. In only one case were there two lesions involving the duodenum and jejunum. The lesion is benign. There has been a report of only one recurrence.

Clinical symptoms usually include abdominal pain due to pyloric outlet obstruction or intussusception if the tumor is located in the small bowel. The lesions can be polypoid or sessile and confined to the submucosa, but they may reach a large size up to 20 cm. Ulceration of the surface is common. The typical histologic features with fibroblasts, eosinophils and scattered chronic inflammatory cells have already been demonstrated. By electron microscopy, these tumors show fibroblasts or myofibroblasts. I would like to have Dr. Simmons show the electron micrographs.

DR. JERRY L. SIMMONS*: The electron microscopic studies demonstrated that this lesion was not of smooth muscle in origin, but that the cells were predominantly fibroblasts. The morphology is that of a typical fibrous inflammatory polyp of the stomach. (Fig. 4)

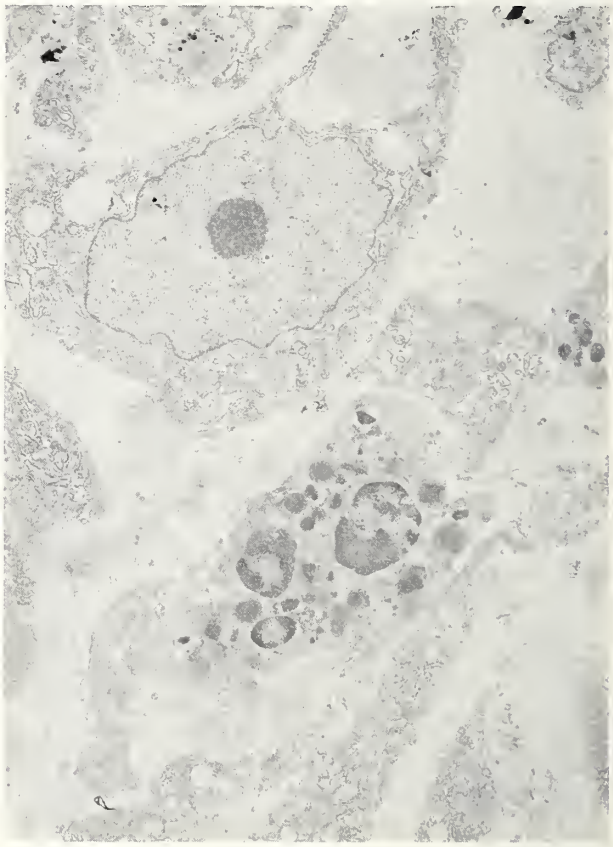

Figure 4
Electron micrograph showing fibroblast (upper cell) and eosinophil (lower cell).

DR. BARLOW: It is important to differentiate this condition of fibrous inflammatory polyp, often called eosinophilic granuloma of the intestinal tract, from other conditions. The lesion has nothing to do with eosinophilic granuloma or histiocytosis X which involves the bone and other tissues. That lesion can also be part of so called Hand-Christian-Schuller or Lettrer-Siwe syndromes which can present as multiple lesions in bone and soft tissue. Histiocytosis X does not usually involve the gastrointestinal tract.

* Pathologist in Chief, Veterans Administration Hospital and Professor of Pathology, School of Medicine, University of South Dakota, Sioux Falls, SD.

The fibrous inflammatory polyps should also be distinguished from eosinophilic gastroenteritis which occurs in the third and fourth decades with a 2-1 male predominence. This condition is often associated with an allergic history or asthma, peripheral blood and tissue eosinophilia. Eosinophilic gastroenteritis may involve the stomach or small bowel with multiple poorly defined areas of eosinophilic infiltration which may lead to stenosis. The condition is responsive to steroids but often recurs. In contrast, inflammatory fibrous polyps are almost always single and rarely recur after resection. There is no peripheral blood eosinophilia, history of allergy or asthma.

DR. LOREN H. AMUNDSON*: The followup on this patient is excellent. The patient is feeling well; the hemoglobin has risen to 11.5 gms and she is eating well. She seems to have no major complaints.

DR. KURT DALLOW†: Why did the patient have fever, chills and an elevated white count?

DR. BARLOW: Probably because of the ulceration of the tumor.

DR. DANIEL HEINEMANN‡: Do you feel that the ulceration of the tumor can explain the gastrointestinal bleeding and does biopsy of a stomach lesion often lead to an appropriate histologic diagnosis?

DR. BARLOW: Yes, the ulceration of the lesion explains the gastrointestinal bleeding. There was a biopsy of this lesion but we did not reach the proper diagnosis until we received the entire specimen. In many cases of adenocarcinoma of the stomach, a diagnosis can easily be made if multiple biopsies are taken. I must agree that on firm lesions such as this lesion or leiomyoma, it may be very difficult to make the diagnosis from a small biopsy specimen.

* Faculty — Family Practice Program, Sioux Falls, SD and Professor of Family Practice, USD School of Medicine, Sioux Falls, SD.

‡ Resident in Family and Community Medicine, Sioux Falls, SD.

† Resident in Family and Community Medicine, Sioux Falls, SD.

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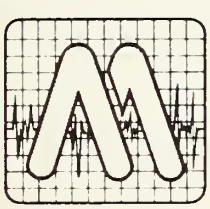

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SD *Auxiliary News*

Legislation
SoDaPAC
Auxiliary Board Meeting
AMA President, Frank Jirka, M.D.

The 1984 SDSMA Auxiliary Legislative Reception and Dinner was held on Monday, Janaury 23, at the Holiday Haus in Pierre.

A group of 31 people consisting of legislators, physicians, and spouses attended the gathering. Also in attendance were Bob Johnson, SDSMA Executive Secretary, and Lorin Pankratz, the new Association lobbyist. District I hosted 100% of their legislators.

The evening was a great success thanks to the endeavors of Mary Cosand, State Auxiliary Legislation Chairman.

On Monday afternoon the SoDaPAC Board of Directors met at the Holiday Haus. N. R. Whitney, M.D., Rapid City, is the 1984-86 chairman. Recommendations for additional board members were submitted at this time.

The Auxiliary State Board held a breakfast meeting at the home of Dr. and Mrs. Marion Cosand on Tuesday, January 23, at 8:30 a.m. On behalf of the Board members I would like to thank the Cosands for their gracious hospitality toward physicians, spouses, and legislators while we were in Pierre.

Following the meeting, Myrna Anderson presented an informative program on real estate investments and property management. Myrna is a licensed realtor in Sioux Falls.

Legislative sessions were attended by some auxiliary members on Tuesday afternoon.

AMA President, Frank Jirka, M.D., presented an informative seminar on Wednesday, Janaury 25, at the Holiday Inn City Center in Sioux Falls. The seminar, entitled "The Future of Medical Practice in a Changing World," was well attended by SD physicians and spouses.

Auxiliary/Medical Society cooperation and interaction has made remarkable strides in recent years in our state. It is important for auxiliary members to be aware of directions in medicine. We are concerned with problems affecting health care and delivery in our country.

Marie Hovland

Marie Hovland, President
South Dakota State Medical Association Auxiliary

SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105

THE COMPETITIVE EDGE

OUTREACH TO PATIENTS

Marketing Glossary (continued)

Positioning — The concept of being in the right place at the right time on a continuing basis, not only in terms of your physical facilities (location) but also in relation to trends in health care delivery, such as extended office hours or prevailing approaches to third-party payers. Positioning involves any fine-tuning adjustment that is designed to put the professional in a commanding position to better serve his market. Patient surveys, community forums, speeches to consumer groups, membership in community clubs, participation in community activities, keeping up with socioeconomic literature in medicine, etc., all are "intelligence-gathering" systems to keep you alert to better ways of "positioning" yourself and your practice.

Pricing — An aspect of marketing that is vital to success. The individual physician operating in the highly codified world of medical practice normally does not have much control over his fees, but rather must conform to the prevailing fee structure. However, some latitude is allowed, particularly in treatment and procedural areas where insurance coverage is ill-defined and, growingly, in innovative approaches to health care delivery stimulated by competition. Judicious attention to fair pricing of such professional offerings could measurably help a physician's public relations posture and the marketing of his practice.

Professional Ethics — A hallmark of the learned professions, particularly medicine and law. In medicine, the ethical component goes back at least as far as ancient Greece and is the basis of the Hippocratic Oath. For years the profession in the United States has been governed by a body of ethics whose watchdog has been the American Medical Association. An extension has been in the statutes in the various states upon which licensing of physicians has been based. Until the late 1970s, AMA and its various components were not challenged in their control of the ethical structure of medical practice. Then, with rapidly rising costs, a litigious era, greater fiscal involvement in health care, and a number of other factors, a movement began in the Federal Trade Commission to limit professional control over advertising by professionals, principally lawyers and physicians as they were the most powerful and highest paid. Advertising and other forms of self-promotion long have been tightly strictured by codes of professional ethics because of their potential to heighten competitive activity and to foster attention to other aspects than client or patient service. Indeed, that is what has happened since the FTC's victory and support by the Supreme Court. The professions are becoming more competitive. However, it is the law of the land, and the prudent physician must survive, if he is to serve his patients at all. That is what this is all about.

Competition — The "buzz" word of the 1980s in medicine, referring primarily to the restructuring of the business aspects of medical practice resulting from (1) more doctors, (2) higher health care costs and growing concern about this at all public levels, (3) economic recession and its impact on third-party payers, including federal and state governments, (4) the FTC ruling's impact on professional control of physician advertising, (5) mounting competitive pressures from alternative health delivery systems (urgent care and convenience health centers), and also from hospitals both public and private engaging in direct health care delivery in various forms, and (6) greater knowledge and sophistication about health matters on the part of a better educated and highly mobile general public. All of these factors and others have converged within the last several years to force practicing family physicians to reorient their professional thinking to encompass more than simply delivering the highest quality medical care to their patients on a comprehensive, continuing basis at the most reasonable price allowing them to make a good living. To help them adjust to this new practice world, the AAFP has devised a judicious, conservative and cost-effective marketing/communications program called "Honing the Competitive Edge." The purpose is to help physicians do a better job of marketing their practices. However, the cardinal precept remains: To deliver the highest quality comprehensive and continuous care to patients, at the most reasonable cost.

July, 1983
Results of the Annual Survey
Family Practice Residency Programs

American Academy of Family Physicians

I. Programs

A. Total Accredited Programs		388
1. Newly accredited programs that have not yet accepted residents		2
B. Program Structure Types		
1. Community hospital based	40	
2. Community based & medical school affiliated	211	
3. Community based & medical school administered	56	
4. Medical school based	64	
5. Military program	17	

II. Residents

A. Total Residents		7,409
1. Total first year residents	2,545	
2. Total second year residents	2,520	
3. Total third year residents	2,344	
B. Total Approved First Year Positions		2,628
C. First Year Fill Rate		97%

D. Increase/Decrease Class Size by Year

	1981-82	*1982-83*	*1983-84*
Class of '84	2,489	2,413	2,344
Class of '85	—	2,578	2,520
Class of '86	—	—	2,545

III. Residency Graduates

A. Total July, 1983 Residency Graduates	2,133
B. Total Graduates from Family Practice Residency Programs Since January 1, 1970	14,967

S D *Future Meetings*

April

The Spectrum of Colon and Rectal Diseases in Primary Care, Hyatt Regency, Minneapolis, MN, Apr. 13-14. Fee: $170. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

May

1984 Conference on Back Pain Rehabilitation, Paper Valley Hotel & Conf. Ctr., Appleton, Wisc., May 7-8. contact: Daria Heyn, OTR, Rehab. Coor., Theda Clark Reg. Med. Ctr., Phys. Med. & Rehab., 130 Second St., Neenah, WI 54956. Phone: (414) 729-2089.

Radiology Update, St. Paul-Ramsey Med. Ctr., St. Paul, MN, May 17-18. 14 hrs. AMA Category I credits. Contact: Charles W. Drage, M.D., Dir. of CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

Topics and Advances in Pediatrics, Malcolm Moos Hlth. Sci. Tower, U. of Minn., Minneapolis, MN, May 17-18. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Medical Radiation Chemistry and Radiopharmaceuticals, MGM Grand, Las Vegas, NV, May 17-20. Fee: $500. 50 hrs. AMA Category I credit. Contact: Nat'l. Instit. for Prof. Ed., 4880 River Bend Rd., Boulder, CO 80301. Phone: (800) 421-8452.

Gynecologic Oncology, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 22. AMA Category I credits. Contact. CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Current Concepts in Radiation Therapy, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 23-25. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Recent Advances in Laboratory Medicine, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, May 30-June 1. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

June

Bariatric Surgery Colloquium, U. of Iowa, Iowa City, IA, June 4-5. AMA Category I credits. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Hepatic, Biliary and Pancreatic Surgery, Willey Hall, U. of Minn., Minneapolis, MN, June 13-16. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Clinical Hypnosis, Earle Brown Ctr., U. of Minn., St. Paul, MN, June 22-23. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Iowa-Western Illinois Neurological Association: Movement Disorders, U. of Iowa, Iowa City, IA, June 22-23. AMA Category I credits. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Intensive Course in Pediatric Nutrition, U. of Iowa, Iowa City, IA, June 25-29. AMA Category I credits. Contact: Richard M. Caplan, M.D., Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Human Aging VII-Senile Dementia, Willey Hall, U. of Minn., Minneapolis, MN, June 27-28. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

OB Ultrasound, Unit F, Hlth. Sci. Bldg., U. of Minn., Minneapolis, MN, June 27-29. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg. 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

The Seventh Annual Black Hills Seminar

The Seventh Annual Black Hills Seminar on Advances in Clinical Pediatrics — June 20, 21 and 22, 1984, at Sylvan Lake Resort, Custer, South Dakota, sponsored by the Department of Pediatrics and Adolescent Medicine, University of South Dakota School of Medicine. Guest faculty include Drs. Frank Oski, John Scanlon, Dan Levin, Robert Vernier and H. David Wilson. For complete conference information contact:

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Published Monthly by the S.D. State Medical Assn.

Volume XXXVII April 1984 Number 4

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Clinicopathological Conference
Fifty-Two Year Old Caucasian Man With Abdominal Pain of Several Days Duration
Cecal Ulcers

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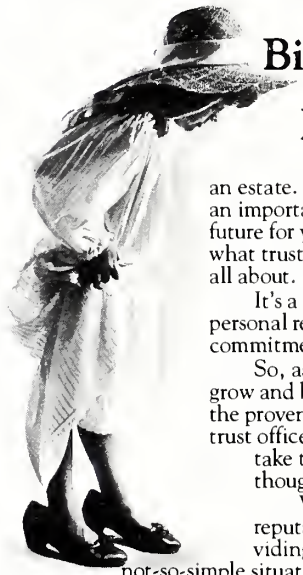

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Official Publication of the
South Dakota State
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the South Dakota Chapter
of The American Academy
of Family Physicians

the South Dakota Chapter
of the American College
of Surgeons

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S D *Medicine*

Blastomycosis of the Tibia: Case Report

H. L. Saylor III, M.D.*
Robert E. Van Demark, Sr., M.D.†
A. E. Hartmann, M.D.‡
L. E. Patterson, Pharm.D.§
Lynn DeMarco, M.D.¶

ABSTRACT

Fungus infection of bone is uncommon. Blastomycosis begins as a primary pulmonary infection, usually mild, which is disseminated to the skin or subcutaneous tissue, and less commonly to the osseous or genito-urinary systems. A case involving the tibia is herein reported with a 6-year followup.

Infection of bone is commonly due to Staph, aureus and other pyogenic organisms. In 1980 the following statement appeared in Campbell's Operative Orthopedic (6th Edition, page 1058) regarding blastomycosis. "It cannot be diagnosed unless suspected." Ordinary cultures of the wound drainage may be entirely negative. A fungus infection should be ruled out in any chronic bone infection. It must be considered in solitary bone lesions of unknown origin, particularly in the tibia where the X-ray appearance can mimic an adamantinoma.

The occurrence of the pulmonary form of North American Blastomycosis in South Dakota has been previously reported in this Journal.[9]

It is appropriate to record the occurrence of this osseous form of the condition in South Dakota together with a case followup of more than 6 years.

CASE REPORT

A white male, age 56, was first seen on January 17, 1977 with the principal complaints of pain and local heat in the upper left tibia. The symptoms began three months previously with no specific injury at that time. He had fallen on New Year's Day and this was reported to increase the symptoms which became steadily worse.

Physical examination revealed an obese white male, 6′2″ tall, weighing 255 pounds. Vital signs were normal. The left tibia showed swelling and tenderness particularly in the region of the tibial tubercle. This mass seemed firm and tender to touch. Examination was not otherwise remarkable, except for two skin lesions posteriorly, one over the upper left scapular area and one over the right lower scapula. These were papular, violaceous with a serpiginous border and measured less than an inch in greatest diameter.

Roentgenographic examination showed an erosive lesion in the bone involving the cortex, (Fig. 1) and minimal degenerative changes in the knee. The differential diagnosis included, besides an infectious lesion, an adamantinoma. The sedimentation rate was only 12 mm per hour Westergren.

On January 18, 1977, an open biopsy was performed through an anterolateral incision. The patient had multiple areas of cortical destruction extending into the medulla and filled with granulation tissue

* Formerly on Orthopedic Clerkship, U.S.D. Medical School; now at Mayo Clinic, Rochester, Minnesota.
† Orthopedic surgeon, 1301 S. Ninth Ave., Sioux Falls, SD
‡ Pathologist, McKennan Hospital, Sioux Falls, SD.
§ Clinical Pharmacologist, McKennan Hospital, Sioux Falls, SD.
¶ Internal Medicine, Leawood, KS, formerly from Sioux Falls, SD.

which was curetted out for biopsy purposes, culture was done and wound closed with hemovac drainage. The patient was sent home awaiting culture and sensitivity report.

Figure 1
Lateral roentgenogram of proximal tibia prior to treatment. Note absence of subperiosteal reaction.

PATHOLOGICAL REPORT

Received from surgery were multiple fragments of grayish-tan to brown curetted soft tissue and bone measuring up to 1.0 cm in greatest dimension. Microscopic examination revealed a mixture of suppurative and granulomatous inflammation with tubercle formation and numerous multinucleated giant cells. Scattered through the cellular reaction were numerous round double contoured single budding yeast forms which with special stains were consistent with Blastomyces organisms (Fig. 2, 3). Fluorescent antibody studies on the tissue and subsequent culture results on Sabourands media, confirmed the diagnosis of Blastomyces dermatitidis.

On identification of the organism, the patient was readmitted to the hospital on February 11, 1977 for intravenous Amphotericin B treatment which he received until February 19, 1977 when he insisted on going home. During his hospitalization he suffered chills, fever, nausea and occasional vomiting as a result of his Amphotericin therapy. His treatment was continued as an outpatient twice weekly for the remainder of his therapy to a total dose of 1490 mg of Amphotericin B. K-lyte® was given to correct a mild hypokalemia (3.2 mEq/liter). Following the completion of his treatment with Amphotericin B, the skin lesions disappeared and his left knee felt better.

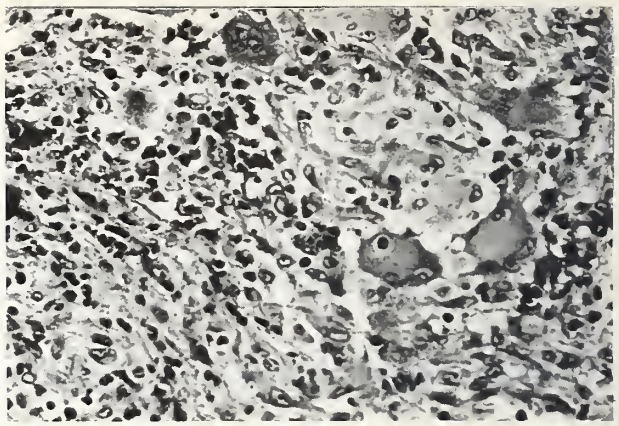

Figure 2
Low power view of granulomatous inflammation with numerous blastomycosis organisms. Hematoxylin and eosin ×105.

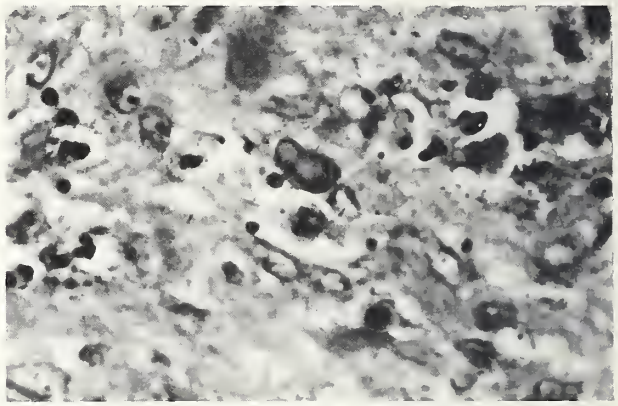

Figure 3
High power view showing typical budding blastomycosis in bone curettings. Periodic acid-Schiff ×430.

The patient's knee remained asymptomatic, and at the time of his subsequent hospital admissions (two) for coronary artery disease he had no complaints of his knee.

The patient was last seen on April 4, 1983. His weight had risen to 270 pounds. His principal complaint was pain in the left hip and knee if he walked on his leg too long. The skin lesions had returned to his back.

The site of his previous surgery showed no evidence of inflammation. The knee joint showed a range of flexion to 20 degrees above a right angle, no effusion, and increased crepitus. His left hip joint showed a positive Patrick-Fabere sign.

Roentgenographic examination showed minimal degenerative arthritis of the left hip and progressive degenerative arthritis of the left knee involving the margins of the patella (Fig. 5) and the medial joint compartment (Fig. 6).

Because of the recurrent skin lesions and the possibility of future knee surgery, the patient was placed on ketoconazole 200 mgs daily. Weight reduction was again strongly encouraged to prevent progres-

sive arthritic changes and insure, if achieved, a better result with surgery.

Figure 5
Lateral roentgenogram knee six years after treatment, with marginal osteophytes about patella.

DISCUSSION

North American blastomycosis is a systemic mycotic infection caused by Blastomyces dermatitidis primarily affecting the lungs or skin. Blastomycosis is prevalent along the Ohio and Mississippi river valleys and in the Mid-Atlantic area. The Blastomyces spores are thought to be prevalent in the soil in these areas and the disease is usually found in people closely associated with outdoor occupations. There is a male preponderance and the majority of cases are in the 20-50 year age group. Inhalation of fungal spores from the soil establishes a primary focus in lung parenchyma and there is rarely dissemination hematogenously to other organ systems. Systemic organs most commonly involved are skin, bone, liver and genitourinary tract. Internists see the disease in its pulmonary form, dermatologists see the cutaneous form of the disease and orthopedists may be confronted with an isolated bone lesion. Bone lesions may be present in 14% to as much as 60% of cases with systemic involvement. When the long bones are involved, there is a tendency towards an epiphyseal location with frequent rupture into

Figure 6
Anteroposterior view of knee showing progressive degenerative changes in the medial joint compartment.

joint spaces. On X-ray, there may be a sharp delineation between the diseased and normal bone; periosteal reaction is rare and bone destruction predominates. The differentiation between tuberculosis and blastomycosis may be difficult, although involvement of the ribs adjacent to the lesions speaks of blastomycosis. Healing is signified by gradual filling in of the bone (Fig. 4). The bones involved with decreasing frequency are the vertebraes, skull, ribs.

Treatment consists of rest of the affected extremity, and splinting of the affected parts; specific treatment formerly was Amphotericin B which must be given intravenously over several hours in increasing doses. The side affects of the drug are significant, including constitutional effects with anorexia, nausea, vomiting, fever, and chills during infusion, hemolytic effects such as anemia, nephrotoxic effects including azotemia, mild proteinuria and hypokalemia.

Because of these reactions to Amphotericin B, we now feel that ketoconazole is the treatment of choice.[3, 7] It is inhibitory to Blastomyces dermatitidis at low concentrations in vitro, and serum levels far exceed minimum inhibitory concentrations for several hours after administration. The usual adult dose is 200 mg in a single daily dose, but up to 400 mg per day can be administered without toxicity.

Figure 4
Lateral roentgenogram of proximal tibia one year after Amphotericin B treatment.

Serum levels should be monitored for dosage adjustment, especially if there has been an inadequate response to the initial dose. Oral therapy should be continued until there is clinical and radiographic evidence that the infection has resolved.

Blastomycosis probably occurs more frequently in this area than it has been recognized on routine cultures. The disease must be diagnosed on the basis of smears and cultures for fungi from the lesion. Oral ketoconazole will resolve the infection and lead to healing of the lesion.

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Before prescribing, see complete prescribing information in SK&F CO. literature or *PDR*. The following is a brief summary.

WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

Supplied: 'Dyazide' is supplied in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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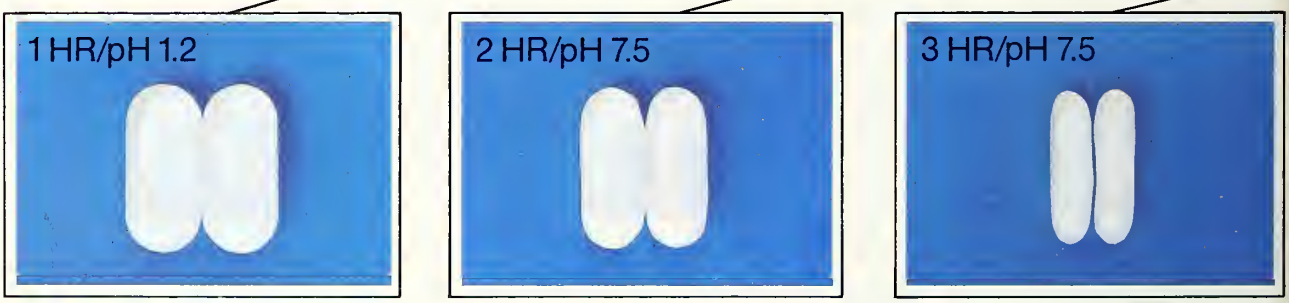

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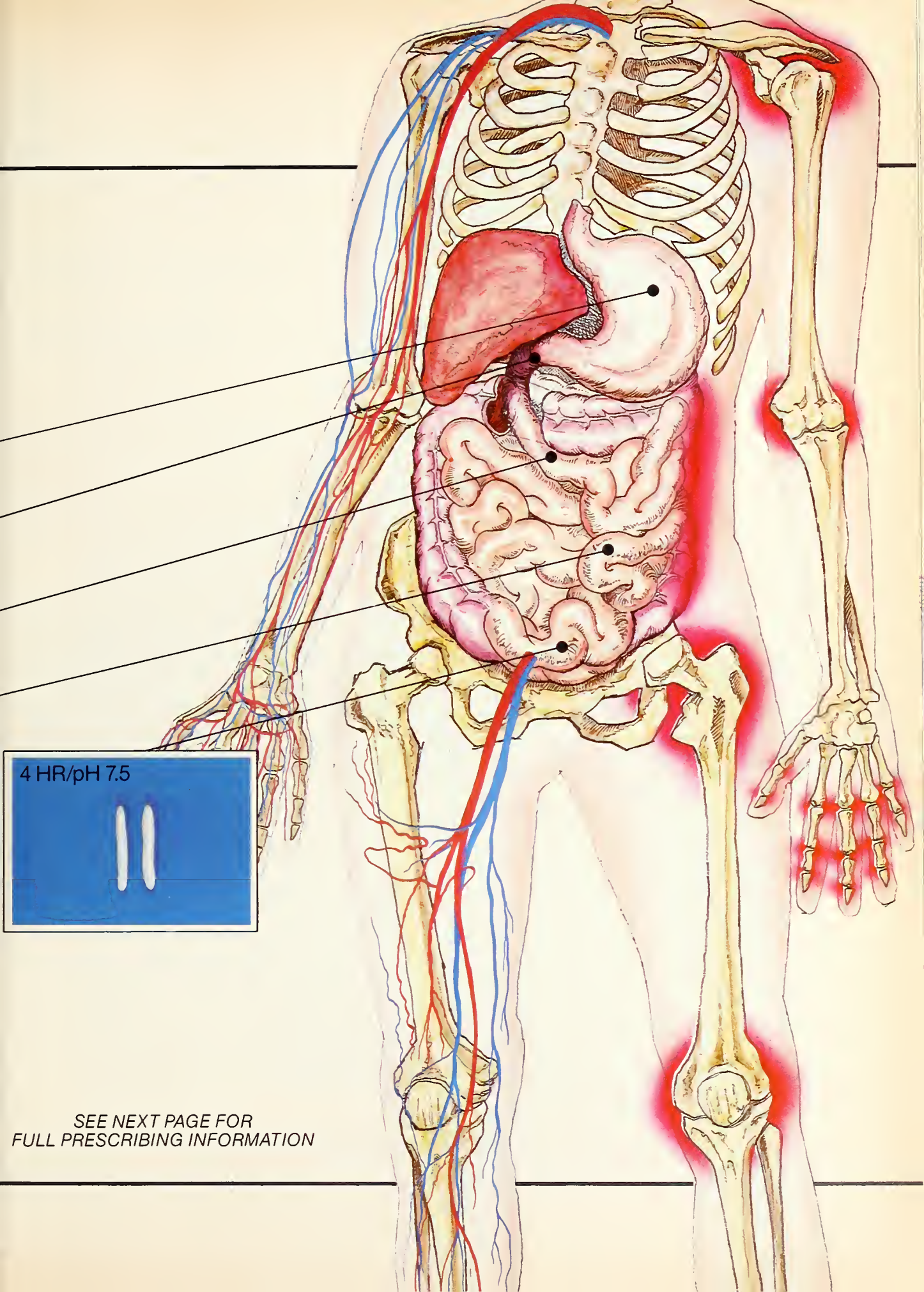
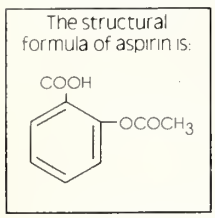


SEE NEXT PAGE FOR
FULL PRESCRIBING INFORMATION

ZORprin (ASPIRIN) Zero-Order Release

DESCRIPTION: Each capsule-shaped tablet of Zorprin contains 800 mg of aspirin, formulated in a special matrix to control the release of aspirin after ingestion. The controlled availability of aspirin provided by Zorprin approximates zero-order release; the *in vitro* release of aspirin from the tablet matrix is linear and independent of the concentration of the drug. □ **CLINICAL PHARMACOLOGY:** Aspirin, as contained in Zorprin, is a salicylate that has demonstrated anti-inflammatory and analgesic activity. Its mode of action as an anti-inflammatory and analgesic agent may be due to the inhibition of synthesis of prostaglandins, although its exact mode of action is not known. □ Zorprin dissolution is pH-dependent. *In vitro* studies have shown very little aspirin to be released in acidic solutions; whereas, Zorprin releases the majority of its aspirin (90%) in a zero-order mode at a neutral to alkaline pH. It is this pH dependence of Zorprin that reduces direct contact between aspirin and the gastric mucosa, resulting in a reduction of its gastrointestinal side-effect potential. □ Bioavailability data for Zorprin have confirmed that plasma levels of salicylic acid and acetylsalicylic acid can be measured 24 hours after a single oral dose. This substantiates a twice daily dose regimen. Multiple dose bioavailability studies showed similar steady-state salicylate levels for Zorprin as for conventional release aspirin using the same total daily dose. Long-term monitoring of salicylate levels showed no signs of accumulation once steady-state levels were reached (4-6 days). □ Studies of *in vivo* prostaglandin levels (PGE2) have shown Zorprin plasma levels of salicylic acid and acetylsalicylic acid to reduce PGE2 levels 14 hours after a single oral 800 mg dose while an equivalent dose of aspirin produced a reduction of PGE2 levels only through six hours. Zorprin's effect on prostaglandins other than PGE2 has not been determined. □ Salicylates are excreted mainly by the kidney, and from studies in humans it appears that salicylate is excreted in the urine as free salicylic acid (10%); salicyluric acid (75%) salicylic phenolic (10%); acyl glucuronides (5%) and gentisic acid (<1%). □ **INDICATIONS & USAGE:** Zorprin is indicated for the treatment of rheumatoid arthritis and osteoarthritis. The safety and efficacy of Zorprin have not been established in those rheumatoid arthritic patients who are designated by the American Rheumatism Association as Functional Class IV (incapacitated, largely or wholly bedridden, or confined to wheelchair, little or no self-care). □ In patients treated with Zorprin for rheumatoid arthritis and osteoarthritis, the anti-inflammatory action of Zorprin has been shown by reduction in pain, morning stiffness and disease activity as assessed by both the investigators and patients. □ In clinical studies in patients with rheumatoid arthritis and osteoarthritis, Zorprin has been shown to be comparable to conventional release aspirin in controlling the aforementioned signs and symptoms of disease activity and to be associated with a statistically significant reduction in the milder gastrointestinal side effects (see ADVERSE REACTIONS). Zorprin may be well tolerated in some patients who have had gastrointestinal side effects with conventional release aspirin, but these patients when treated with Zorprin should be carefully followed for signs and symptoms of gastrointestinal bleeding and ulceration. □ Since there have been no controlled trials to demonstrate whether or not there is any beneficial effect or harmful interaction with the use of Zorprin in conjunction with other nonsteroidal anti-inflammatory agents (NSAI), the combination cannot be recommended (see **Drug Interactions**). □ **Because of its relatively long onset of action, Zorprin is not recommended for antipyresis or for short-term analgesia.** □ **CONTRAINDICATIONS:** Zorprin should not be used in patients known to be hypersensitive to salicylates or in individuals with the syndrome of nasal polyps, angioedema, bronchospastic reactivity to aspirin, renal or hepatic insufficiency, hypoprothrombinemia or other bleeding disorders. Zorprin is not recommended for children under 12 years of age; it is contraindicated in all children with fever accompanied by dehydration. □ **WARNINGS:** Zorprin should be used with caution when anticoagulants are prescribed concurrently, since aspirin may depress platelet aggregation and increase bleeding time. Large doses of salicylates may have hypoglycemic action and enhance the effect of the oral hypoglycemics, concomitant use therefore is not recommended. However, if such use is necessary, dosage of the hypoglycemic agent must be reduced. The hypoglycemic action of the salicylates may also necessitate adjustment of the insulin requirements of diabetics. □ While salicylates in large doses have a uricosuric effect, smaller amounts may reduce water excretion and increase serum uric acid. □ USE IN PREGNANCY: Aspirin can harm the fetus when administered to pregnant women. Aspirin interferes with maternal and infant hemostasis and may lengthen the duration of pregnancy and parturition. Aspirin has produced teratogenic effects and increases the incidence of stillbirths and neonatal deaths in animals. □ If this drug is used during pregnancy, or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus. □ Aspirin should not be taken during the last 3 months of pregnancy. □ **PRECAUTIONS:** Appropriate precautions should be taken in prescribing Zorprin for patients who are known to be sensitive to aspirin or salicylates. Particular care should be used when prescribing this medication for patients with erosive gastritis, peptic ulcer, mild diabetes or gout. As with all salicylate drugs, caution should be exercised in prescribing Zorprin for those patients with bleeding tendencies or those on anticoagulants. □ In order to avoid exacerbation of disease or adrenal insufficiency, patients who have been on prolonged corticosteroid therapy should have their therapy tapered slowly rather than discontinued abruptly when Zorprin is made a part of the treatment program. □ Patients receiving large doses of aspirin and/or prolonged therapy may develop mild salicylate intoxication (salicylism) that may be reversed by dosage reduction. □ Salicylates can produce changes in thyroid function tests. □ Salicylates should be used with caution in patients with severe hepatic damage, preexisting hypoprothrombinemia, Vitamin K deficiency and in those undergoing surgery. □ Since aspirin release from Zorprin is pH dependent, it may change in those conditions where the gastric pH has been increased as a result of antacids, gastric secretion inhibitors or surgical procedures. □ **Drug Interactions:** (See **WARNINGS**) Aspirin may interfere with some anticoagulant and antidiabetic drugs. Drugs which lower serum uric acid by increasing uric acid excretion (uricosurics) may be antagonized by the concomitant use of aspirin, particularly in doses less than 2.0 grams/day. Nonsteroidal anti-inflammatory drugs may be competitively displaced from their albumin binding sites by aspirin. This effect may negate the clinical efficacy of both drugs. Also, the gastrointestinal inflammatory potential of nonsteroidal anti-inflammatory drugs may be potentiated by aspirin. The combination of alcohol and aspirin may increase the risk of gastrointestinal bleeding. □ Aspirin may enhance the activity of methotrexate and increase its toxicity. □ Sodium excretion produced by spironolactone may be decreased in the presence of salicylates. Concomitant administration of other anti-inflammatory drugs may increase the risk of gastrointestinal ulceration. Urinary alkalinizers decrease aspirin's effectiveness by increasing the rate of salicylate renal excretion. Phenobarbital decreases aspirin's effectiveness by enzyme induction. □ **Pregnancy Category D.** See **WARNINGS** Section. □ **Nursing Mothers:** Salicylates have been detected in the breast milk of nursing mothers. Because of the potential for serious adverse reactions from aspirin in nursing infants, a decision should be made whether to discontinue nursing or discontinue the drug, taking into account the benefit of the drug to the mother. □ **ADVERSE REACTIONS: Hematologic:** Aspirin interferes with hemostasis. Patients with a history of blood coagulation defects or receiving anti-coagulant drugs or with severe anemia should avoid Zorprin. Aspirin used chronically may cause a persistent iron deficiency anemia. □ **Gastrointestial:** Aspirin may potentiate peptic ulcer, and cause stomach distress or heartburn. Aspirin can cause an increase in occult bleeding and in some patients massive gastrointestinal bleeding. However, the greatest release of active drug from Zorprin is designed to occur in the small intestine over a period of time. This has resulted in fewer symptomatic gastrointestinal side effects. □ **Allergic:** Allergic and anaphylactic reactions have been noted when hypersensitive individuals have taken aspirin. Fatal anaphylactic shock, while not common, has been reported. □ **Respiratory:** Aspirin intolerance, manifested by exacerbations of bronchospasm and rhinitis, may occur in patients with a history of nasal polyps, asthma, or rhinitis. The mechanism of this intolerance is unknown but may be the result of aspirin-induced shunting of prostaglandin synthesis to the lipoxygenase pathway and the liberation of leukotrienes, *e.g.* slow-reacting substance of anaphylaxis. □ **Dermatologic:** Hives, rashes, and angioedema may occur, especially in patients suffering from chronic urticaria. □ **Central Nervous System:** Taken in overdoses, aspirin provides stimulation which may be manifested by tinnitus. Following initial stimulation, depression of the central nervous system may be noted. □ **Renal:** Aspirin rarely may aggravate chronic kidney disease. □ **Hepatic:** High doses of aspirin have been reported to produce reversible hepatic dysfunction. □ **OVERDOSAGE:** Overdosage, if it occurs, would produce the usual symptoms of salicylism: tinnitus, vertigo, headache, confusion, drowsines, sweating, hyperventilation, vomiting or diarrhea. Plasma salicylate levels in adults may range from 50 to 80 mg/dl in the mildly intoxicated patient to 110 to 160* mg/dl in the severely intoxicated patient. An arterial blood pH of 7.1 may indicate serious poisoning. The clearance of salicylates in children is much slower than adults and should receive due consideration when aspirin overdosages occur in infants; salicylate half-lives of 30 hours have been reported in infants 4-8 months old. Treatment for mild intoxication should include emptying the stomach with an emitic, or gastric lavage with 5% sodium bicarbonate. Individuals suffering from severe intoxication should, in addition, have forced diuresis by intravenous infusions of sodium bicarbonate and dextrose or sodium lactate. In extreme cases, hemodialysis or peritoneal dialysis may be required □ (*A plasma salicylate level of 160 mg/dl in an adult is usually considered lethal.) □ **DOSAGE & ADMINISTRATION:** *In order to achieve a zero-order release, the tablets of Zorprin should be swallowed intact.* □ *Breaking the tablets or disrupting the structure will alter the release profile of the drug.* □ *It is recommended that Zorprin be taken with sufficient quantities of fluids (8 oz. or more).* □ **Adult Dosage:** For mild to moderate pain associated with rheumatoid arthritis and osteoarthritis, the recommended initial dose of Zorprin is 1600 mg (2-800 mg tablets) twice a day. Because of Zorprin's prolonged release of aspirin into the bloodstream, Zorprin tablets may be taken as a b.i.d. dose. Further adjustment of the dosage should be determined by the physician, based upon the patient's response and needs. Since it will take 4-6 days to reach steady-state levels of salicylic acid with Zorprin, it is recommended dosages be given for at least one week before further adjustment. In general, patients with rheumatoid arthritis seem to require higher doses of Zorprin than do patients with osteoarthritis. □ **Zorprin is not recommended for children below the age of 12.** □ **HOW SUPPLIED: Zorprin Tablets 800 mg;** plain, white capsule-shaped tablets. □ Bottles of 100 Tablets—NDC 0524-0057-01. □ **Caution:** Federal law prohibits dispensing without prescription. □ U.S. Patent No. 4,308,251. □ **Manufactured and Distributed by: BOOTS PHARMACEUTICALS, INC., Shreveport, Louisiana 71106 U.S.A.**



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D
Clinicopathologic Conference

Fifty-Two Year Old Caucasian Man With Abdominal Pain of Several Days Duration

Timothy M. Peterson, M.D.*
Discusser

John F. Barlow, M.D.†
Editor

CASE #1 005 444 (83-3508)

This 52-year old Caucasian male entered Sioux Valley Hospital with the chief complaint of abdominal pain of several days duration.

The patient had been well until three days prior to admission when he developed periumbilical episodic crampy pain associated with vomiting but without hematemesis. He had noted darkening of the urine over the two weeks prior to admission but had not noticed dysuria, change in bowel habits, or in color of the stools or any other genitourinary or gastrointestinal symptoms. At the time of admission, the pain had increased but was not associated with chills or fever. The patient had had a normal bowel movement the previous evening.

Three months prior to admission he had been admitted for abdominal pain of unknown etiology which had resolved. At that time the 12-panel chemistry, urinalysis and hemogram were within normal limits.

Past medical history revealed that the patient had had a subtotal colectomy for ulcerative colitis with an anastomosis between the ileum and rectum. He had been relatively asymptomatic but had been followed closely since this operation with sigmoidoscopies. The patient had a history of cerebral palsy causing spastic weakness in both the upper and lower extremities. There was no other significant symptoms elicited on review of systems. The patient did have an allergy to penicillin. Other than Metamucil®, the patient took no medication.

PHYSICAL EXAMINATION: Pulse 76 per minute and regular, respirations 20 per minute and regular, blood pressure 110 systolic and 60 diastolic, temperature 37°C, weight 69 kilograms, height 170 cm. The patient appeared to be in some distress. There was a question of icterus of the skin and conjunctivae. Examination of the head and neck was otherwise unremarkable except for miotic pupils. The chest was clear to auscultation and percussion. The heart was not enlarged and there was a regular rhythm with no murmurs. Examination of the abdomen showed no palpable abdominal or costovertebral angle tenderness. No palpable organs or masses were noted in the abdomen. There was mild periumbilical tenderness. Rectal examination was negative and there was no stool obtainable for guaiac testing. The extremities had bounding equal pulses and were unremarkable. The neurologic examination showed spasticity of the upper and lower extremities and no abnormalities of the cranial nerves.

LABORATORY DATA: Urinalysis — amber, hazy, specific gravity 1.028, pH 5.0, negative for protein, glucose, hemoglobin, 3+ ketones, positive bilirubin; sediment 0-1 white cell/hpf, no red cells or epithelial cells, calcium oxalate crystals and uric acid crystals were present in moderate amounts. Hemoglobin 15.0 gm/dl, hematocrit 45 vol% with normal red cell indices. White count 5.9 $\times$ 10^9/liter with 81% segmented neutrophils, 1% neutrophilic bands, 16% normal lymphocytes, 1% monocytes and 1% basophils. The red cells were normochromic normocytic on smear. The platelet count was 290 $\times$ 10^9/liter. A prothrombin time and partial thromboplastin time were within normal limits. Blood urea nitrogen, uric acid, cholesterol, total protein, phosphorus, creatinine, amylase, sodium, potassium, chloride, and lactic dehydrogenase (LD) were within normal limits. Alkaline phosphatase was 362 IU/L (normal 0-115 IU/L). Asparate aminotransferase 175 IU/L (normal 0-60 IU/L). Total bilirubin 3.7 mg/dl, direct bilirubin 2.6 mg/dl and indirect bilirubin 1.1 mg/dl. Calcium 8.3 mg/dl (normal 8.4 to 10.7 mg/dl), glucose 133 mg/dl. An electrophoretic pattern was within normal limits. A chest film was negative. An electrocardiogram was within normal limits. A gallbladder ultrasound was read as normal. A radionuclide cholescintigraphy revealed nonvisualization of the gallbladder after four hours. The biliary tract was patent with flow of material into the bile duct within 90 minutes. An operation was performed.

* Resident in Family and Community Medicine: Affiliated with USD School of Medicine, Sioux Falls, SD.
† Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital: Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

DR. PETERSON: This patient entered with abdominal pain, an elevated total bilirubin, and direct bilirubin and an elevated alkaline phosphatase. This certainly suggests a cholestatic process. I will approach this case as a differential diagnosis of obstructive jaundice with direct hyperbilirubinemia.

The first major category would be conditions causing intrahepatic cholestasis. The major entities in this category include Dubin-Johnson syndrome, Rotor's syndrome, drug cholestasis, and primary biliary cirrhosis. Dubin-Johnson's hyperbilirubinemia is seen in young individuals with chronic intermittent jaundice. It is familial and is associated with an elevated alkaline phosphatase. Needle biopsy is diagnostic demonstrating a brown lipofuscin pigment in the liver. Rotor's syndrome is very similar except there is no brown pigment in the liver and there is a normal alkaline phosphatase. Both the above syndromes are rare and have a good prognosis with no progressive liver disease. They are inborn errors of the metabolism of bile pigments at the cellular level.

Many drugs can cause intrahepatic cholestasis. Probably the most common is chlorpromazine. However, other phenothiazines, arsphenamine, paraminosalicylic acid, chlorpropamide, nitrofurantoin, methyltestosterone and other anabolic steroids can produce a similar problem.

One must mention primary biliary cirrhosis, but this disease occurs predominantly in middle-aged females and is an autoimmune process. Eighty percent of these patients have a positive test for serum antimitochondrial antibody. We are not given this information in this case. These patients usually present with progressive pruritis, malaise, fatigue, increasing icterus, and eventually develop signs of portal hypertension secondary to cirrhosis. The syndrome may be associated with symptoms of mixed connective tissue disease, rheumatoid arthritis, thyroid disease or Sjorgren's syndrome. In addition to the high antimitochondrial antibody, these patients have an elevated IgM, an elevated alkaline phosphatase and cholesterol and other abnormal liver function tests. Liver biopsy shows an elevated copper level as well as a histologic pattern with initially portal inflammation and granuloma formation progressing to bridging fibrosis, loss of bile ducts and eventuating in an end stage cirrhotic picture. The life expectancy of these patients is 5-10 years in symptomatic patients and over 10 years in asymptomatic patients. The condition can be treated with penicillamine or azathioprine with variable results.

Other conditions such as chronic active hepatitis and chronic persistent hepatitis may give this pattern of liver function tests seen. Chronic active hepatitis shows a distinct histologic pattern and has a much worse prognosis than does the chronic persistent pattern. Patients with alpha 1 antitrypsin deficiency, hemochromatosis, sarcoidosis or amyloidosis can present the pattern of results seen in this case, although rarely. We are not given any information such as serum iron or ferritin levels or determination of alpha 1 antitrypsin to support these diagnoses. Wilson's disease is an inborn error of copper metabolism which may produce abnormal liver function tests. There are no clinical or laboratory data given to support this diagnosis. One must also consider toxic chemicals such as phosphorus, carbon tetrachloride or other agents as well as infectious conditions such as ascending cholangitis, pylephlebitis, leptospirosis, infectious mononucleosis and syphilis. These infectious diseases are unlikely as the patient presented with no fever. Lymphoma is always a possibility either producing intrahepatic cholestasis from hepatic metastases or extrahepatic cholestasis with pressure on the biliary system by involved lymph nodes.

The other major category of disease beside intrahepatic cholestasis is, of course, extrahepatic cholestasis. If the patient had cholelithiasis with or without acute cholecystitis, a stone may have passed into the common duct causing obstruction (choledocholithiasis). This rather common sequence of events could easily explain this patient's symptoms. Often ascending cholangitis will result from partial obstruction. In this case, ascending cholangitis is unlikely because the patient did not have fever and chills or bacteremia secondary to this condition. Pancreatitis or pancreatic neoplasm could cause obstruction of the common bile duct as could a bile duct neoplasm. Carcinoma of the gallbladder is frequently associated with cholelithiasis and may have spread to the bile ducts causing obstruction. This condition certainly could give the scan picture seen in this case. External pressure by enlarged lymph nodes or tumor such as I described above with lymphoma could produce this. A choledochal cyst could produce this condition but I would expect it to have been mentioned on the cholescintigraphic study.

I would like to seriously consider the condition of primary sclerosing cholangitis. This entity produces progressive obstructive jaundice in the absence of calculi or previous surgery. There is generalized thickening and stenosis of the biliary ductal system. The same condition may be caused by a sclerosing malignancy, but primary sclerosing cholangitis by definition means that there is no malignant disease. This condition can be associated with ulcerative colitis, regional enteritis, or retroperitoneal fibrosis. The etiology of the condition is unknown. Cultures of these ducts have been negative but secondary bile acid produced by bacterial degenerative products has

been postulated. An autoimmune cause in those cases associated with regional enteritis, ulcerative colitis, vasculitis, and Sjorgren's syndrome has been postulated. The condition also has been thought to be part of a generalized fibromatosis and has been associated with retroperitoneal fibrosis, Reidel's fibrosing thyroiditis, pseudotumor of the orbit, sclerosing mediastinitis, relapsing pancreatitis and immunodeficient states. Patients who have sclerosing cholangitis have a male predominance and are usually in the third to fifth decade. They present with painless jaundice, pruritis, and nonspecific upper abdominal discomfort associated with hepatomegaly and splenomegaly. I have already mentioned the common association with ulcerative colitis. It has been estimated, however, that less than 1% of patients with ulcerative colitis will develop primary sclerosing cholangitis.

Patients with primary sclerosing cholangitis often have an elevated direct bilirubin, alkaline phosphatase and serum transaminase as well as eosinophilia. Serologic markers such as antimitochondrial antibody, antinuclear antibody or antibodies to smooth muscle are usually negative. The lupus erythematosus (LE) preparation and tests for HBsAG are usually negative. Oral cholecystography and intravenous cholangiography, ultrasonography, and computer tomography may not detect the sclerosed ducts. Percutaneous transhepatic cholangiography is often helpful and the most helpful diagnostic procedure is often endoscopic retrograde cholangiography. Often primary sclerosing cholangitis is not diagnosed until exploratory laparotomy. The liver shows no specific abnormality, although there may be a variety of changes in the portal tract on histologic examination of the liver biopsy. The gallbladder may show chronic granulomatous inflammation or papillary hyperplasia of the mucosa. Biopsy of the bile ducts shows fibrosis and chronic inflammation.

Patients with sclerosing cholangitis primarily affecting the distal common bile duct, or occurring after an attack of acute necrotizing cholangitis, often have a good prognosis with the majority surviving seven to ten years. Patients with chronic diffuse sclerosing cholangitis have a mean survival of six years and those with chronic diffuse sclerosing cholangitis associated with inflammatory bowel disease have a mean survival of only 4.5 years. Patients have been treated with steroids, cholestyramine, and vitamin supplements (A, D, and K) and calcium with some success. Surgery can benefit some patients with distal disease. Penicillamine has also been used in this disease.

In summary, this patient had obstructive jaundice. He certainly could have had gallstones or a neoplasm involving the head of the pancreas, bile ducts or gallbladder to explain this condition. I am going to stick my neck out and say the patient had primary sclerosing cholangitis.

Dr. Timothy Peterson's Diagnosis
Primary Sclerosing Cholangitis

DR. BARLOW: I would like to show the radionuclide cholescintigram (gallbladder and biliary scan) in this case. Early images after the injection of disofenin show good visualization of the liver. The intrinsic hepatic ducts and common bile duct are seen at the 20 and 60 minute views but no radionuclide has entered the duodenum. In the normal scan, radionuclide should enter the duodenum within 60 minutes. By 90 minutes, material is seen in the duodenum. This indicates that there is partial obstruction in the common duct. More important, the gallbladder is not visualized on any of the views including delayed views up to 4 hours. In the normal situation the gallbladder should be visualized within one hour. The absence of visualization in the gallbladder is reasonably diagnostic of obstruction of the cystic duct. This is indicative of acute cholecystitis.

In summary, there is a pattern of complete cystic duct obstruction (acute cholecystitis) and partial common duct obstruction with delay of radionuclide appearance in the duodenum, although this delay can be seen in normal patients due to spasm of the sphincter of Oddi. (Figs. 1, 2)

Figure 1
Liver, biliary tree are visualized but gallbladder is not seen in expected area (arrow).

Figure 2
Radionuclide appears in intestinal tract after some delay indicating functionally patent biliary system but gallbladder is still not visualized.

The surgery was performed by Dr. Peter J. O'Brien.* A right subcostal incision was made. The gallbladder was encased in omentum and acutely inflamed. A purse-string suture was placed in the fundus of the gallbladder and the gallbladder was aspirated. The bile was very light colored, almost white, suggesting hydrops of the gallbladder. A gram stain indicated no bacteria. The cystic duct was dilated and there was a soft stone very tightly impacted at the cystic duct-common duct junction. Because the patient did have jaundice and because the surgeon did not want to push this stone into the common duct from the cystic duct, a common duct exploration was performed. The wall of the duct was thick and the actual lumen of the common duct was minute. It was very difficult to locate the lumen of the common duct distally. There were several stones in the common duct which were removed. A choledochoscope was passed with difficulty and no stones were seen but the duct was narrowed. The choledochoscope could not be passed into the distal common duct but a scoop was passed, although not all the way to the ampulla. No stones were revealed. A small size 10 T-tube was placed in the duct and a series of cholangiograms were performed. The bile ducts were all extremely small. A frozen section biopsy of the material from the common duct was called benign, mild fibrosis.

* General Surgeon, Sioux Valley Hospital, Faculty USD School of Medicine, Sioux Falls, SD.

In the pathology laboratory we received a gallbladder measuring 6 x 3 x 2 cm. There were multiple irregular yellow calculi 0.1 to 0.5 cm. Sections of the wall of the gallbladder revealed ulceration in the mucosa and both acute and chronic inflammatory cells associated with focal hemorrhage. (Fig. 3) Also submitted were fragments of calculi from the common duct and a biopsy of the common duct showing mild fibrosis.

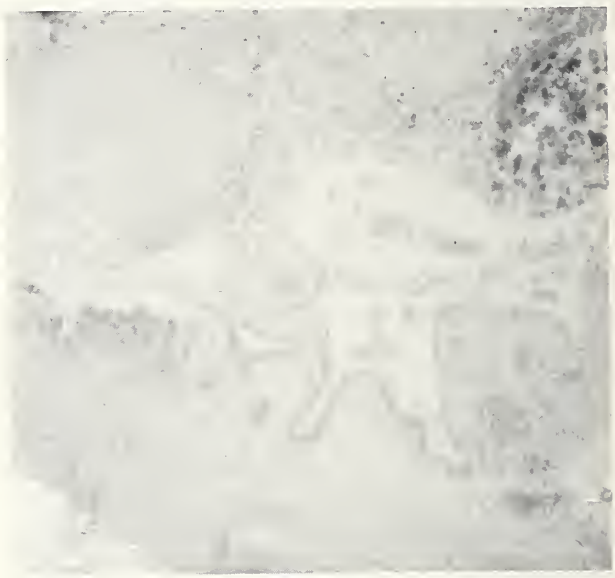

Figure 3
Gallbladder showing acute ulceration of mucosa with overlying segmented neutrophils which also infiltrate wall below mucosa.

This patient's pathologic picture is a rather complex one. Certainly the patient had cystic duct obstruction and probably partial common duct obstruction demonstrated both by the cholescintigram and the fact that there was a stone impacted in the cystic duct as well as common duct stones. In addition, the patient has sclerosing cholangitis as Dr. Peterson has suggested.

FINAL ANATOMIC DIAGNOSES

1. **CYSTIC DUCT OBSTRUCTION WITH ACUTE AND CHRONIC CHOLECYSTITIS AND CHOLELITHIASIS**
2. **CHOLEDOCHOLITHIASIS**
3. **SCLEROSING CHOLANGITIS**

I would now like to review what I feel is an important new functional test that adds to the armamentarium in diagnosing conditions such as gallbladder disease and abnormalities of the biliary duct systems — radionuclide cholescintigraphy.

There have been a number of different methods for imaging the liver, biliary tract and gallbladder promoted in the last decade, e.g. computer tomography, ultrasonography, radionuclide liver scanning and radionuclide cholescintigraphy. One fruitless approach has been to try to determine which of these techniques is best and use it for all situations. As might be expected, all of these techniques are quite useful and may offer a noninvasive, sensitive method depending on what answers are being sought. I will attempt to point out in this short dissertation the place of radionuclide cholescintigraphy.

I would like to say at the onset, cholescintigraphy is an exciting new noninvasive technique which can give information not available through the use of other modalities. However, it is not a substitute for ultrasonography, computer tomography or radionuclide liver scanning.

WHAT IS CHOLESCINTIGRAPHY?

Although radionuclide visualization of the liver and biliary system has been available through the use of 131 iodine labelled Rose Bengal, the advent of immunodiacetic acid (IDA) compounds labelled with 99m technetium offers imaging agents secreted by the liver which consistently and adequately visualize the gallbladder and bile ducts as well as the liver. Cholescintigraphy must be performed after a 4-6 hour fast or nonvisualization of the gallbladder may occur. A 3-5 millicurie dose of 99Tc IDA is injected intravenously. We prefer the diisopropyl derivative called DISIDA or disofenin because adequate scans can be obtained even with high bilirubin levels. This dose gives a minimal radiation exposure equivalent to a chest film. Anterior views are taken immediately and approximately every 10 minutes up to one hour with one lateral view at 45 minutes. If the gallbladder and biliary tract are visualized within one hour and radionuclide is seen within the duodenum, the scan may be considered to be within normal limits. If the gallbladder or duodenum are not visualized, delayed views for up to four hours must be performed.

WHAT INFORMATION DOES CHOLESCINTIGRAPHY PROVIDE?

Since the clinical presentation of the acute abdomen due to acute cholecystitis can be confused with other surgical and nonsurgical conditions, cholescintigraphy provides the clinician with an accurate, noninvasive method for preoperative diagnosis of acute cholecystitis.

Cholescintigraphy is the most sensitive method for determination of the presence or absence of patency of the cystic duct and, hence, the diagnosis of acute cholecystitis. No other method reliably provides this information. If the gallbladder is not visualized after delayed views up to four hours, cystic duct obstruction is almost a certainty (98-99%). Since acute cholecystitis is usually due to a stone lodged in the cystic duct, the diagnosis of cystic duct blockage and acute cholecystitis are almost synonymous. It is interesting that the rare cases of acalculus cholecystitis usually show functional cystic duct obstruction with nonvisualization of the gallbladder.

COMMON PATTERNS OF CHOLESCINTIGRAPHY

1. **Normal** — The gallbladder, hepatic ducts, common ducts and duodenum are visualized within 60 minutes, confirming cystic duct and common duct patency.
2. **Acute cholecystitis** — Gallbladder does not visualize even up to four hours but the common duct and duodenum visualize within 60 minutes. This is cystic duct obstruction.
3. **Common duct obstruction** — Despite good hepatic visualization, the gallbladder, hepatic and common duct and duodenum do not visualize within 60 minutes or longer. Acute cholecystitis may or may not be present.
4. **Chronic or occasionally acute cholecystitis** — Gallbladder visualizes after 1 hour but within 4 hours. The biliary tree and duodenum visualize within 60 minutes. This pattern is probably due to partial cystic duct obstruction from edema, debris or fibrosis.
5. **Nonspecific, chronic cholecystitis, ampullitis** — The gallbladder and nondilated common duct preferentially visualize but the duodenum is not visualized within 60 minutes. This is probably due to functional common duct obstruction and may be due to spasm or fibrosis of the sphincter of Oddi region. Some feel that sincalide, the octadecapeptide of cholecystokinin, cholecystokinin itself or a fatty meal may produce gallbladder emptying in this situation.

WHAT THEN ARE THE ADVANTAGES OF CHOLESCINTIGRAPHY?

1. Rapid method for assistance in evaluation of the acute abdomen.
2. Low radiation dose.
3. Demonstrates functional patency of cystic and common duct even in early obstruction when methods depending on detection of dilatation of the biliary tree as indicative of obstruction are unreliable, or in cases where functional patency is present in the face of a dilated biliary tree.
4. Serendipity
   a. The liver is well visualized and hepatic abnor-

malities may be detected.
   b. The radionuclide is also excreted in the urine so renal or urinary tract abnormalities may be detected.
   c. Intestinal abnormalities may be detected after the radionuclide enters the intestine.
5. Noninvasive
6. No serious reactions to intravenous radionuclide compounds.

WHAT ARE THE DISADVANTAGES OF CHOLESCINTIGRAPHY?

1. Poor anatomic resolution. Determination of ductal dilatation or visualization of stones or neoplasm within the biliary tree is not always accomplished.
2. Prolonged fasting as in hyperalimentation may cause nonvisualization of gallbladder. The injection of sincalide prior to the procedure may obviate this problem.
3. **Cholescintigraphy does not reliably detect cholelithiasis.**
4. Although delayed visualization of the gallbladder suggests chronic cholecystitis, cholescintigraphy is not a reliable method for the diagnosis of chronic cholecystitis.
5. In congenital biliary atresia, cholescintigraphy at the present time is only a fair diagnostic method. Longer halflife compounds such as ruthenium 99 may be more efficacious.

EVALUATION OF THE POSTOPERATIVE PATIENT VIA CHOLESCINTIGRAPHY

Symptoms due to continuing abnormalities in the biliary system after cholecystectomy are not uncommon and include retained stones in the common duct, bile duct stricture or cystic duct remnant. The following cholescintigraphic patterns are helpful, but it must be remembered that delayed views are always necessary.

1. Visualization of the bile ducts and duodenum within 60 minutes rules out functional obstruction.
2. Dilated ducts noted by other techniques may demonstrate functional patency by cholescintigraphy.
3. Partial or complete common duct obstruction can be demonstrated.
4. Cholescintigraphy is the most sensitive method for detecting a cystic duct remnant. The problem is that evaluating the significance of a cystic duct remnant is difficult.
5. Occasionally photon deficiency within the biliary tree will represent a retained stone.
6. A bile leak can be detected.

EVALUATION OF BILIARY ENTERIC BYPASS VIA CHOLESCINTIGRAPHY

1. Although this is not a common indication for cholescintigraphy, the functional patency and presence of a biliary fistula can be reliably determined.
2. Evaluation of afferent or efferent loop obstruction after Billroth II gastrectomies can also be demonstrated.

COMPARISON OF CHOLESCINTIGRAPHY WITH OTHER TECHNIQUES OF IMAGING

1. **Ultrasonography**
   This is a very reliable technique for detection of cholelithiasis and biliary ductal dilatation. It is noninvasive and gives no radiation. For these reasons, it is a very valuable tool but it does not compare in reliability to cholescintigraphy in the detection of acute cholecystitis.
2. **Oral Cholecystography**
   This is a time honored, reliable technique for detection of cholelithiasis. However, it cannot detect acute cholecystitis reliably. It also does not reliably determine biliary tree patency. There is often a minimum 12-hour delay with this procedure and nonvisualization of the gallbladder is a nonspecific finding.
3. **Intravenous Cholangiography**
   This technique has been used to determine abnormalities of the biliary tree. It can detect biliary duct dilatation well and visualize filling defects but it cannot detect acute cholecystitis reliably, determine functional patency of the biliary tree in all instances, and is associated with serious reactions to the intravenous contrast agents — morbidity 10-20%, mortality 1%, due to anaphylaxis, nausea, vomiting, bronchospasm, pulmonary edema.

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HISTORY OF THE BLACK HILLS WINTER SKI SEMINAR

At the end of the February 2-4, 1984 meeting, nearly 900 practicing physicians, family practice residents, medical students, and allied health personnel have attended nine Black Hills Winter Ski Seminars, all held at the Holiday Inn of the Northern Black Hills in Spearfish.

The first Black Hills Winter Ski Seminar was held January 29-31, 1976. This meeting resulted from the vision of local Black Hills area Academy Members. They developed the format for the meeting, still in use, which includes early morning and early evening scientific sessions, allowing the mid-portion of the day for winter sports activities. They enlisted the aid of the South Dakota Academy of Family Physicians to obtain prescribed credit for the meeting and help with conduct of the scientific sessions. The early involvement of these physicians helped insure a solid base for this annual event, now to enter its tenth year.

Practicing physicians attending these Black Hills Winter Ski Seminars have had access to 113 prescribed hours of AAFP credit. The scientific sessions have been presented by a total of 53 speakers, two of them family physicians. Family Practice Residents from the Sioux Falls program have been present at all nine seminars, and medical students from USDSM have been present at most of the annual events. SDAFP Family Practice Club events have been a part of some of the seminars. Five AAFP staff persons from the national office in Kansas City have been guests of SDAFP during these years.

Persons attending these seminars have enjoyed winter sports activities in the scenic Black Hills, including alpine and cross-country skiing. Reports from the slopes and trails have included several aching joints and muscles, however, only one sprained ankle and no other more serious injuries.

Scientific sessions have provided a group learning experience for SDAFP members and guest registrants, the content offering a continuing exposure to the identified core of knowledge in family practice. The objectives have been to provide scientific educational units which offer a review and present new concepts so that the knowledge level of the physician may allow early recognition of these disorders, management if possible, and an awareness of the indications for consultation and referral for secondary and tertiary care as appropriate. Content areas of these nine seminars have included: ENT; Emergency Medicine; Maxillofacial Injuries; Medicine, including segments in Cardiology (including preventive risk factors, videotape laboratories in pacemaker insertion and Swan-Ganz catheter placement), Diabetes Mellitus, Gastroenterology, Hematology, Obesity, Oncology, and Pulmonary Medicine; Musculoskeletal Disorders, including Arthritis; Neurology; Ophthalmology; Orthopedics; Pediatrics, including Pediatric Radiology; Psychiatry and Behavioral Medicine; and Sports Medicine, including taping and strapping sessions.

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For nearly a hundred years, the Statue of Liberty has been America's most powerful symbol of freedom and hope. Today the corrosive action of almost a century of weather and salt air has eaten away at the iron framework; etched holes in the copper exterior.

On Ellis Island, where the ancestors of nearly half of all Americans first stepped onto American soil, the Immigration Center is now a hollow ruin.

Inspiring plans have been developed to restore the Statue and to create on Ellis Island a permanent museum celebrating the ethnic diversity of this country of immigrants. But unless restoration is begun now, these two landmarks in our nation's heritage could be closed at the very time America is celebrating their hundredth anniversaries. The 230 million dollars needed to carry out the work is needed now.

All of the money must come from private donations; the federal government is not raising the funds. This is consistent with the Statue's origins. The French people paid for its creation themselves. And America's businesses spearheaded the public contributions that were needed for its construction and for the pedestal.

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President's Page

Since 1939 physicians have struggled against the dangers in proposed federal medical legislation. American Medical Association president, Dr. Donovan Ward, referred to this in 1964 when he said to the House of Delegates of the American Medical Association, "The patient is the sufferer when the quality and quantity of care depends on budgetary considerations rather than on medical need." He predicted that federal health programs proposed in 1965 would impose costly and burdensome controls upon hospitals which would determine admissions and discharge of patients, and he prophesied that the taxes required "to cover the enormous and unpredictable cost of hospitalization would endanger the entire Social Security system; the economic protection of millions of Americans over 65." Dr. Ward was correct, and he echoed the voices of many of us.

Politicians, not physicians, designed the Social Security Act of 1965. It is flawed by inadequate financing, extravagant guarantees for health services, and the wrong incentives for providers of health care. All of these have contributed to the spiraling costs of the Medicare and Medicaid programs, but as very knowledgeable Senator Durenberger of Wisconsin states, "Half of the increases can be attributed to improved technologies, and the public wants them."

Physicians cannot control public appetites. We will need to resist the onslaught of political proposals intended to rescue the ailing programs which will surface in the current session of the Congress. In the words of AMA president, Dr. Frank Jirka, Jr., "If our profession allows (the) challenges before us to splinter it, who will stand for physicians and for our patients?"

Joseph N. Hamm, M. D., President
South Dakota State Medical Association

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Cecal Ulcers

Albin J. Janusz, M.D., F.A.C.S.*

ABSTRACT

This is a case presentation of a cecal ulcer which presented as an acute abdomen. Cecal ulcers are an uncommon occurrence. They are difficult to diagnose and are usually asymptomatic with eventual perforation. Treatment is surgical and differential diagnosis from carcinoma of the cecum is difficult.

The occurrence of solitary nonspecific ulcers of the colon was first described in 1832 and for a number of years remained only an interesting observation usually made at post-mortem examinations. In the past four decades the lesion has been documented to be a definite clinical and pathological entity. However, the frequency of this nonspecific ulceration is unknown and the number of cases reported in the last 140 years suggests either that it is an uncommon entity or that many cases are resolved without diagnosis.

The following is a case presentation of a solitary cecal ulcer which presented as an abdominal mass.

CASE PRESENTATION

A 29-year-old white female was seen because of fever, abdominal cramps, nausea and the presence of an abdominal mass. She gave a history of about three to four weeks of nonspecific abdominal cramps and nausea. After being seen by her physician and no specific physical findings present, she was treated with Librax® and a low residue diet. The patient did well until six days after her examination when she developed chills, nausea and right lower quadrant pain. Examination of the abdomen revealed no distention, but with depressed bowel sounds and with tenderness in the right lower quadrant and rebound, and a palpable tender mass. Rectal examination supported the findings of a mass in the right lower quadrant. Flat plate of the abdomen showed an ill defined mass in the right lower quadrant, probably an abscess. The patient presented with a temperature of 101.6°F., hemoglobin of 13 g/dl, WBC 9,300/MM^3 with 1% monos, 22% lymphs, 62% segs and 15% stabs. The urinalysis was normal.

Because of the mass and because of the patient's symptoms, it was felt that the patient probably had a ruptured appendix with an abscess and she was scheduled for surgery.

At surgery she was found to have a mass at the ileo-cecal junction with purulent material being expressed from the mesenteric border of the ileo-cecal valve. Because of the mass, it was felt that a right hemicolectomy was indicated. This was performed and postoperatively the patient did well. The pathology report was that of a resected cecum and ascending colon with normal ileum. The colon was found to have a large mural abscess secondary to mucosal ulceration and perforation. There was no evidence of neoplasia. The appendix was normal.

The patient had an uncomplicated postoperative course and was discharged.

DISCUSSION

The occurrence of idiopathic, isolated ulcers of the colon, exclusive of rectal ulcers, number slightly more than 150 cases in the world literature. Many theories have been proposed to explain the etiology of nonspecific isolated colon ulcers but none have been proven. Constipation with subsequent trauma resulting in a stercoral ulcer does not explain the frequent occurrence of cecal ulcers in the fluid en-

* Clinical Assistant Professor of Surgery, USD School of Medicine, Aberdeen, SD.

vironment of the cecum. A number of authors have considered ischemia to be important but none have been able to develop convincing proof of this hypothesis regardless of the location of the ulceration of the bowel.

Thrombosed blood vessels are sometimes seen in the submucosa, but the question remains as to the primary or secondary role of thrombosis. There has been some correlation found between intravascular coagulation in patients, localized colon ulcers and fibrin microvascular thrombi beneath the ulcers. Fibrin microthrombi may then represent the final event in the pathogenesis of discrete colon ulcers.

Colon ulcers have mimicked acute intestinal obstruction, diverticulitis, appendicitis and carcinoma. Perforation of these ulcers is as high as 80%. Bleeding has also been reported at about 15-16% in some cases and as high as 28% in some series. The mortality has been reported in Mark and Bollingers series as 35%.

Diagnosis by x-ray barium enema is nonspecific as well as by angiography. Colonoscopy may have some benefit but it is difficult to identify bleeding discrete ulcers with this technique.

The clinical presentation of nonspecific ulcers of the colon can be quite varied. In general, most cases occur in the fourth and fifth decades of life, although some patients in their teens have been reported. The incidence is greatest in males. Patients with ulcerations in the cecum or ascending colon usually present with right lower quadrant abdominal pain and nausea, but without vomiting. Fever and leukocytosis may be present. There are no pathognomonic signs to aid in the diagnosis of cecal ulcers.

Presence of gross blood in the stool is uncommon in patients with cecal ulcers. The usual initial diagnosis is appendicitis, although pelvic inflammatory disease, ovarian disease, regional enteritis, and tuberculosis enteritis may be considered. Palpable tender right lower quadrant mass, as seen in this case, is often present in the latter two conditions and not in uncomplicated cecal ulcers. Perforation of the cecum with peritonitis is not uncommon. These ulcers usually perforate on the mesenteric border in contrast to diverticula which occur along the antimesenteric border. Discrete colon ulcers have been reported recently in end-stage renal disease. The relationship to end-stage renal disease has been obscure.

TREATMENT

The conventional management of discrete colon ulcers has been surgery with local excision or partial colectomy. Many surgeons will perform a partial colectomy for an ulcerated mass which resembles carcinoma. Simple drainage in the case of perforated colon ulcers appears to be associated with a high mortality rate. Some authors advocate colotomy and biopsy and antibiotic therapy with good results. A few cases have been followed with successful nonoperative management. The majority of cases will require laparotomy and partial colectomy for the safest and best results.

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Attention: Medical Students, Resident Physicians, and Spouses

The SDSMA Auxiliary is looking for spouses in your special membership category. A recent issue of HORIZONS has been sent to all AMA resident physician and medical student members. You were asked to take this issue home to your spouse to help spread the word about the AMA Auxiliary and all it has to offer as a support group for medical families.

HORIZONS is a newsletter meant specifically for resident physicians' and medical students' spouses. If you know of one interested in receiving HORIZONS contact our state Medical Student Spouse Advisor, Karen Fletcher, RR 2, Vermillion, 57069, (624-8337), or our Medical Student Spouse Liaison, Jane Solberg, RR 1, Baltic, 57003, (543-5245).

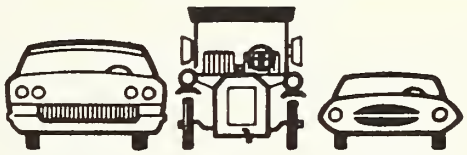
AMA Auxiliary President Glenda Bates addressed the AMA's Resident Physician and Medical Student Sections at their meetings held in conjunction with the AMA's Interim House of Delegates meeting. Mrs. Bates clarified the role of the AMA Auxiliary and detailed the benefits that the organization can offer to spouses of resident physicians and medical students.

Two important videotape programs are now available from the AMA's Division of Practice Management. "Borrowing Money" is produced specifically for resident physicians planning to set up a practice. "Developing a Marketing Plan for Your Medical Practice," introduces the physician to the concepts of marketing as they apply to a medical practice. To order, contact Gail Simpson, Marketing Services, AMA, 535 N. Dearborn St., Chicago, Ill. 60610; 312/751-5961.

A SPECIAL WELCOME to all State Medical Association and Auxiliary members to our annual meeting in Rapid City, June 7-10, 1984. Mary Johnson and Jackie Slingsby are co-chairmen of auxiliary activities and Mary Butz is District 9 president. Co-hosts are Districts 1 and 6. Their respective presidents are Barb Bunker, Aberdeen and Shirley Berry, Mitchell.

Marie Hovland

Marie Hovland, President
South Dakota State Medical Association Auxiliary



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NEED FOR PRIMARY CARE PHYSICIANS IN SOUTH DAKOTA

Compared to other states, South Dakota has consistently ranked at or near the bottom in the supply of professionally active physicians.[1, 2] By definition, professionally active physicians include all active, licensed physicians (M.D.s and D.O.s) in private practice or residency training, and federal and state employed physicians.

In the early 1970s, there was much concern about the State's ability to attract physicians. In 1973, there were an estimated 600 active physicians in the State, or about 8.8 per 10,000 persons.[3] One study showed that of the 508 physicians in private practice in 1973, 179 would reach or exceed the age of 65 by 1983.[4] Since there had been no net increase in physicians in the State since 1960, there was cause for concern in having an adequate future supply of physicians.

In the last ten years, the number of physicians in South Dakota has risen quite dramatically. As of November, 1983, there were an estimated 900 professionally active physicians, or about 12.9 per 10,000 persons. This amounts to a fifty percent increase in the last ten years! It seems reasonable to assume that most of this increase has been in the primary care specialties which include family practice, internal medicine, obstetrics/gynecology, and pediatrics; or general practice.

Even with the increase in physician supply, certain communities and institutions have shortages of primary care physicians. Some areas are viable private practice sites. Other areas, such as those located on Indian reservations, are not, at least from an economic sufficiency standpoint.

The State Department of Health undertook a brief assessment of need for primary care physician services in December, 1983. One area of interest concerned physician replacement at age 65. Although physicians do not necessarily quit practicing upon reaching this age, it was felt that replacement should at least be considered once a physician reaches this age. Table I shows communities which are projected to have nonfederal, primary care physicians in practice who are at, or above age 65. Only those towns which are projected to have one or more physicians at or above age 65 are shown. As the table shows, based solely on age, the State will need an additional 102 primary care physicians by 1990.

Table I

Projected Vacancies in the Number of Nonfederal Primary Care Physicians, Based on Retirement Age of 65

Community	Number Nonfederal Primary Care Physicians Active Practice 1984	At or Over Age 65 by 1985[1]	Reaching Age 65 Between 1986 and 1990[2]
Sioux Falls	96	8	13
Rapid City	43	6	2
Huron	15	7	0
Watertown	17	3	4
Aberdeen	25	5	1
Yankton	22	3	2
Sturgis	8	3	1
Hot Springs	6	2	2
Brookings	10	1	2
Mitchell	14	2	1
Chamberlain	3	2	0
Milbank	2	1	1
Freeman	4	2	0
Deadwood	4	1	1
Redfield	4	0	2
Tyndall	2	0	1
Arlington	1	1	0
Kimball	1	1	0
Belle Fourche	3	0	1
Platte	2	0	1
Wagner	3	1	0
Armour	1	1	0
Phillip	2	0	1
Miller	3	0	1
Menno	1	1	0
Parkston	3	1	0
Tripp	1	0	1
Wessington Springs	2	1	0
Madison	5	0	1
Canton	2	0	1
Salem	1	1	0
Eureka	2	1	0
Britton	2	0	1
Colton	1	1	0
Dell Rapids	4	1	0
Valley Springs	1	1	0
Lemmon	2	1	0
Centerville	1	1	0
Marion	1	1	0
Total	320	61	41

[1] Birthdate on or before 1920
[2] Birthdate between 1921 and 1925

Bernard L. Osberg
Willis F. Stanage, M.D.
Lawrence J. Massa
South Dakota State Department of Health

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1. The Supply of Health Manpower, 1970 Profiles and Projections to 1990. DHEW Publication No. (HRA) 75-38. Bureau of Health Resources Development, U.S. Department of HEW, December, 1974, pp 28-29.
2. Physician Distribution and Medical Licensure in the U.S., 1978. Department of Statistical Analysis, American Medical Association, Chicago, 1980.
3. Directory of Physicians and Physical Therapists Licensed in South Dakota. South Dakota State Board of Medical and Osteopathic Examiners, January, 1973. Unpublished data provided also by a survey of state and federal health care institutions located in the State.
4. The Erosion of Rural Physician's Services in South Dakota, 1940-1983. Comprehensive Health Planning Program, State Department of Health, 1973.

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Obituaries

Arthur J. Barrett, M.D., Rapid City, died October 10, 1983, at the age of 50. He was born October 13, 1932, at Mapleton, Iowa. He received his medical degree from Creighton University Medical School, Omaha, Nebraska in 1962 and interned at Sacred Heart Hospital, Spokane, Washington 1962-63. He established his practice in Rapid City in 1963 and practiced there until his death.

Dr. Barrett was an active member of the South Dakota State Medical Association. He served as councilor from 1975-1982; as secretary of the Ninth District from 1974 until his death; he was a member of the SoDaPAC Board; and at the time of his death, he was Speaker of the House.

Dr. Barrett is survived by his wife, Mary; and his children.

Richard W. Honke, M.D., of Wagner died November 7, 1983, at the age of 69. Dr. Honke was born March 30, 1914 in Butte, Nebraska. He received his medical degree in 1938 from Creighton University Medical School in Omaha, Nebraska. He served in the Army Medical Corps from 1942 to 1946 and was awarded a Purple Heart. He began his practice in Wagner in 1946 and practiced there until his death.

Survivors include his wife, Florence; a son, Dr. Richard Honke, Wagner; a daughter, Dr. Sandra Honke, Rapid City; three grandchildren; and two sisters.

Dr. Robert R. Donahoe, 63, a former Sioux Falls surgeon died recently in Orlando, Fla. Dr. Donahoe was born Feb. 11, 1920 in Sioux Falls. He graduated from Notre Dame University in 1942 and Georgetown Medical School in 1945. He interned at Providence Hospital, Washington, DC in 1946. He then returned to Sioux Falls where he practiced surgery for 25 years. Along with his father, Dr. Stephen Donahoe, his brother, Dr. John Donahoe and Dr. G. I. W. Cottom, they founded the Donahoe Clinic now the Central Plains Clinic.

He married Delores Drabek in 1951. They moved to Orlando eight years ago, where Dr. Donahoe practiced surgery.

Survivors include his wife; a son, Daniel; three daughters, Rosemary, Nancy and Carol, all of Orlando; a brother, Dr. John W. "Jack" Donahoe and a sister, Mrs. Ann Scholten, both of Sioux Falls.

SD Future Meetings

May

Cardiac Pacing, Sheraton-Midway, St. Paul, MN, May 4. CME credits available. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Allergy and Clinical Immunology, Mayo Mem. Aud., Minneapolis, MN, May 10-12. Fee: $225. 16½ hrs. AAFP & AMA Category 1 credits. Contact: CME, Box 293 Mayo Mem. Bldg. 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

American Geriatrics Society's 41st Annual Scientific Meeting, Denver Hilton Hotel, Denver, CO, May 15-18. Fee: $335. CME Credits available. Contact: Linda Hiddemen Barondess, Ex. VP, Am. Geriatrics Society, 10 Columbus Cir., New York, NY 10019. Phone: (212) 582-1333.

June

33rd Annual Session of the University of Utah School on Alcoholism and Other Drug Dependencies, U. of Utah, Salt Lake City, UT, June 17-22. AMA Category I credits available. Contact: U. of Utah School on Alcoholism & Other Drug Dependencies, P. O. Box 2604, Salt Lake City, UT 84110. Phone: (801) 533-7087.

31st Annual Family Practice Review, Estes Park, CO, June 11-16. Contact: Off. of Postgrad. Med. Ed., U. of Colo. School of Med., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

The Seventh Annual Black Hills Seminar on Advances in Clinical Pediatrics, Sylvan Lake Resort, Custer, SD, June 20-22. Contact: Lawrence Wellman, M.D., Prog. Coor., USD School of Med., P. O. Box 5039, Sioux Falls, SD 57117-5039. Phone: (605) 333-7178.

July

20th Annual Internal Medicine Program, Estes Park, CO, July 9-13. Contact: Off. of Postgrad. Med. Ed., U. of Colo. School of Med., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

Aspen Summer Skin Seminar, Aspen, CO, July 10-14. Contact: Off. of Postgrad. Med. Ed., U. of Colo. School of Med., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

Dynamic Psychotherapy, Aspen, CO, July 30-Aug. 3. Contact: Off. of Postgrad. Med. Ed., U. of Colo. School of Med., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

August

Black Hills Summer Seminar, sponsored by SD Chapter AAFP, Howard Johnson Motor Lodge, Rapid City, SD, Aug. 9-11. AAFP & AMA Category 1 credits. Contact: Jan Anderson, SDSMA, 608 West Ave., N., Sioux Falls, SD 57104. Phone: (605) 336-1965.

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Volume XXXVII May 1984 Number 5

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Somatopsychic Disease at the South Dakota Human Services Center

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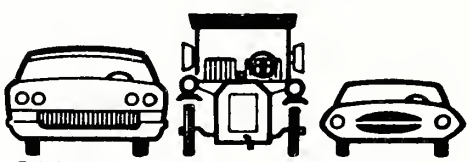

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BRIEF SUMMARY

PRDCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina), PRDCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PRDCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PRDCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PRDCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCAROIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCAROIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with light aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCAROIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCAROIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%; palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCAROIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCAROIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCAROIA therapy, has been reported twice in the extensive world literature.

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Volume XXXVII/No. 5 May 1984 (ISSN 0038-3317)

Official Publication of the
South Dakota State
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the South Dakota Chapter
of The American Academy
of Family Physicians

the South Dakota Chapter
of the American College
of Surgeons

the South Dakota
Psychiatric Association
of the American
Psychiatric Association

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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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Somatopsychic Disease at the South Dakota Human Services Center

David A. Smith, M.D.*

ABSTRACT

The medical literature suggests that the incidence of nonpsychiatric disease with psychiatric symptomology presenting to psychiatric institutions is fairly high.[3-5] Incidence of somatopsychic illness at SDHSC is not statistically evaluated but appears to be low, yet further awareness of the possibility of physical disease presenting with psychiatric symptoms is clearly indicated. Cases are discussed.

Somatopsychic illness refers to "physical" disease that masquerades as "psychiatric" disease.

At first brush we all have fairly clear ideas of what constitutes "physical" illness and "psychiatric" illness yet these ideas are probably more arbitrary than we realize. A low serum sodium from SIADH that results in delerium would clearly be a "physical illness" with a "psychiatric" presentation. But, then, are the various psychoses if caused by derangements of neurotransmitters really neuro-endocrine diseases and, therefore, physical illnesses?

Following this line of thought we destroy the arbitrary but useful distinction between "physical" and "psychiatric" illness. For purposes of this discussion we refer to "physical" illnesses (and therefore somatopsychic illnesses) as diseases most appropriately diagnosed and treated by physicians other than psychiatrists and "psychiatric" illnesses as diseases with which psychiatrists have special expertise.

The incidence of physical illness in psychiatric patients at the SDHSC, as elsewhere,[1, 3, 4, 8] is large and, in many, physical illnesses complicate or worsen the co-existing psychiatric illnesses for which these patients are hospitalized.

An occasional patient is referred and admitted, however, who is later found to have a physical cause for the psychiatric symptomatology for which they have been referred to SDHSC.

CASE 1

B.F. was a 59 year old Caucasian female committed involuntarily to the SDHSC in transfer from the psychiatric unit of a private hospital. She had been residing at a community nursing home until her admission to that psychiatric unit following suicide attempts (strangling herself with oxygen tubing and stabbing herself in the neck rather severely with a fork). Furthermore, she exhibited helplessness, and demanding and highly manipulative behaviors.

She was a known insulin dependent diabetic, had multiple allergies and severe chronic obstructive lung disease, asthmatic type. She carried diagnoses of CHF and HBP. She had complaints of vague abdominal discomforts and was anorexic. The patient presented to SDHSC bedridden and appearing so infirm that she could not be appropriately placed on our acute psychiatric unit but instead was sent to the acute care hospital.

Chest x-rays showed cardiac enlargement and bilateral pleural effusions. KUB was normal. Bilateral hand x-rays were normal. Upper GI normal. Electrocardiogram showed ST changes of digitalis or ischemia and possible old anterior MI. Later chest x-rays showed improvement and later EKGs showed atrial fibrillation/flutter. SMA12 showed calcium 10.7 mg/dl, phosphorus 5.4 mg/dl, glucose 205 mg/dl, BUN 57 mg/dl, but with other parameters normal.

* Associate Professor, Department of Family Medicine, USD School of Medicine; joint appointment, Department of Psychiatry; Chief of Medicine, South Dakota Human Services Center, Yankton, SD.

Sodium was 137 mEq/L, potassium 2.3 mEq/L, and creatinine 1.2 mg/dl. Digoxin level was 1.4 ng/ml. UA showed 25-30 WBC's/hpf., 3+ glucose, 2+ protein and culture grew E. coli. Hb was initially 10.0 grams with Hct 31.9% and indices were microcytic, hypochromic. Reticulocyte count 2.9%, Aminophylline level 14.5 ng/ml. TSH was 3 uIu/ml. ABGs showed pO_2 62, pCO_2 40, pH 7.45 on room air. Serial calcium determinations were from 10.0 to 12.3 mg/dl. Stool for blood negative.

After this initial evaluation and adjustment of medications the patient was returned to SDHSC pending results of parathormone assay sent to reference laboratory. A calcium level of 11.3 mg/dl (N = 8.5-10.6) had a corresponding PTH level of 2486 pg/ml (N = 0-500).

The patient was re-admitted to the acute care hospital. IVP showed small calcifications in the upper calyx of left kidney. Abdominal sonograms were normal. After further preoperative screening a near total parathyroidectomy was performed and the pathologist reported hyperplasia of four parathyroid glands.

The day following surgery the patient was noted to have had a remarkable change in behaviors. She was less demanding, less manipulative, more alert and no longer appeared depressed. Her calcium levels were slightly subnormal. She returned to SDHSC for milieu therapy to further reduce her passive aggressive and passive dependent traits and was eventually discharged to a community home as a trial step toward returning to her own home. She was discharged with DSM Axis II diagnosis of Passive Dependent Personality Disorder and Axis III diagnoses of IDDM, COPD-C, Hyperparathyroidism — status post operative, hypertension, and multiple allergies.

CASE 2

A 58 year old Caucasian female presented for voluntary admission to SDHSC with a one year history of mental problems including dependency, feelings of worthlessness and depression and suicidal ideation which seemed to post-date her husband's retirement from his job. In the three months preceding this admission she began to refuse to eat, care for herself or to walk. She worried that she might lose her legs. She was a known diabetic and attempted a suicide by taking an overdose of Diabinese®.

She was examined by an endocrinologist and a neurosurgeon who found no physical reason for her inability to walk, although she was found to be hypokalemic. She was on no diuretics. She then began seeing a psychiatrist who eventually transferred the patient to SDHSC with a diagnosis of severe depression. On admission to SDHSC the examining psychiatrist noted the inability to walk but no other physical abnormalities. Stool for blood was negative, FBS 336 mg/dl, sodium 140 mEq/lt, potassium 3.2 mEq/lt. Urine showed 1+ albumen, 2% clinitest, 2+ blood, 18-24 WBCs/hpf, 2-4 RBC/hpf, many bacteria. Hb was 14.0 with Hct 39.5. White count 10,700. TB skin test was negative. BUN was 10.3 mg/dl and creatinine 0.8 mg/dl. A repeat sodium was 142 and potassium 2.7 mg/l.

Because of the patient's extreme flaccid paresis the psychiatrist made consultations with Family Medicine and Neurology. On physical exam HEENT was normal, thyroid not palpable, chest was clear and breasts normal, heart regular rhythm without murmur at rate of 72/min. Abdomen was normal to palpation, had surgical scars consistent with known hysterectomy and cholecystectomy. Truncal obesity and purple stria were noted. Genital exam and vaginal exam were normal noting blind vaginal cuff. Rectal exam revealed poor tone, and flaccid paresis was noted on musculoskeletal exam. Ecchymoses were seen near the right eye and on her arms. She was disoriented to time but oriented to place and person and her mood was quite depressed. The following laboratory tests were then obtained: Creatinine clearance 46 ml/min (but poor total volume), AM cortisol greater than 50 mcg/dl and PM cortisol greater than 50 mcg/dl. (N = 5-25); CSF protein 56 mg/dl (N = 15-45), CSF glucose 194 mg/dl (N = 45-90 mg/dl), no cells in CSF, and VDRL non-reactive; 17 ketosteroids 35.2 mg/24h (N = 5-15) and 17 ketogenic steroids 25.0 mg/24h (N = 5-14).

The patient was sent to Sacred Heart Hospital for further evaluation. Repeat 17 ketogenic and ketosteroids and diurnal cortisols were similar to those above. Chest x-rays showed a left hilar mass. A dexamethasone suppression test was begun but the patient suffered a cardiac arrest during that day and though initially resuscitated died a few hours later.

Autopsy revealed carcinoma, bronchogenic, left lung, large cell undifferentiated. (Though unproven, this tumor probably was hormone producing and the cause of a Cushing's syndrome.) Metastatic carcinoma to liver, retroperitoneal nodes, right lung perihilar nodes, adrenal glands, thyroid, and pancreas. Edema, lungs, marked. Infarct left lung, small. Fat necrosis, pancreas and pericolic adipose tissue. Subarachnoid hemorrhage — small. Atherosclerosis, generalized. Hypertrophy of heart. Absence uterus, cervix, gall bladder and appendix. Diabetes mellitus, clinical dx. The patient had no DSM Axis I or Axis II diagnoses attributed at death.

CASE 3

S.L., a 40 year old Caucasian male was committed to SDHSC from a county jail for what was

believed to be "malingering with potential for self harm." The patient, a known insulin dependent diabetic, was found guilty of petty theft (siphoning gas) and was sentenced to four months in a county jail. After a few days there he began complaining of headache behind the right eye, of weakness of his left arm and of slurred speech. He reported losing control of his left arm and that "it won't do what I want it to." He reported jerking movements of his left hand and arm. He was taken to the area hospital and a normal physical exam was recorded. A diagnosis of psychosomatic reaction was given and related to the "stress" of being incarcerated. He was returned to jail but the following day he vomited. He was again taken to the hospital, seen by a second physician, and diagnosed as "malingering." A CAT scan was attempted but the patient moved during the exam and it was of "unacceptable quality for diagnostic purposes." He again returned to jail. He was noted to stumble, almost fall while showering and later was committed to SDHSC for "malingering behavior that would produce self injury."

On admission to SDHSC the patient was noted to be fully oriented but had slowed speech, poor eye contact, BP was 130/92. Funduscopy was not possible due to small but equal pupils. Chest and heart exams were normal. Abdomen normal. Neurological exam revealed cranial nerves II-XII intact, tongue deviated slightly to the left. DTR's were symmetrical and normal. Pinprick was intact. Muscle strength normal and symmetric. Fine motor coordination was decreased in the left hand. Romberg was normal, however, gait was slow and he performed heel to toe walk with great difficulty.

Additional history was obtained of a motor vehicle accident in which he sustained a fractured jaw nine years earlier and that he had been hit with a rifle butt in the face and left occiput during apprehension for his petty theft. He did not lose consciousness with that head trauma.

While at SDHSC the patient's diabetes was monitored and, although it required some adjustment, was not severely out of control. Localized left sided seizures were observed. An EEG showed an abnormal sleep record with right hemisphere slowing. CAT scan showed a localized right frontal lobe lesion consistent with posttraumatic contusion (hemorrhagic infarct). Skull x-rays were negative. Neuropsychiatric testing revealed impairment of function associated with right temporal and sensory motor areas with the dysfunction appearing acute rather than chronic. MMPI showed the patient to be "emotionally distant from others." "Self defeating acting out in criminal fashion would be predicted." Verbal IQ was 105 while performance IQ was only 82, a difference suggesting right hemisphere impairment.

The patient was placed on Dilantin® and when therapeutic blood levels were obtained was discharged as not mentally ill. He was given a DSM Axis II diagnosis of anti-social personality disorder and Axis III diagnoses of insulin dependent diabetes mellitus and encephalopathy, focal, right frontal lobe, post-traumatic contusion, contrecoup type with left focal seizures.

DISCUSSION

A great number of conditions including disease states, neoplasms, nutritional deficiencies, toxins, and pharmacologic agents may produce psychiatric symptomatology sometimes as the first or only symptom of a disease.[2-6] Since many of these conditions are unusual they may be easily overlooked. Furthermore, the time consuming and unpleasant aspects of psychiatric problems often interfere with careful history taking, physical examination and possible medical workup by the primary care physician and hasten referral of the patient to a psychiatrist before it is really determined that this is the appropriate specialty to receive the referral. McIntyre and Romano[5] point out that psychiatrists may be poorly equipped to pick up the patient mistakenly referred to them who actually has a somatopsychic illness, although the psychiatric staff at SDHSC has seemed quick enough to identify that these patients presented here had something other than psychiatric illness. Many somatopsychic conditions are easily diagnosed by primary care physicians if they are entertained in the differential diagnosis.

CONCLUSION

Psychiatric symptoms, e.g., anxiety, depression, hallucinosis, etc., do not have one specific cause. A variety of psychiatric conditions may cause each. Likewise, any physical illness affecting the neurochemistry may cause each. We humans have only a few basic behaviors to express our very complex physiology.

We have presented an array of these in an attempt to demonstrate the non-specific nature of somatopsychic illness. Statistical evaluation has not been done since we made no attempt to define our population, deal with the frequent re-admissions to this hospital, or to find other possible cases of somatopsychic illness dealt with by other members of the SDHSC staff, referred elsewhere instead of admitted, etc. There seems to be little need to generate more statistics on this subject after the work of Hall et al., and Koranyi[1,3,4,6] but only to rekindle an awareness of somatopsychic illness. We wish also to gain understanding of the frequency of somatopsychic illness presenting to the SDHSC from our

referring physicians. The incidence appears to be low indicating good awareness of these possibilities by those who make referrals to SDHSC. Still, vigilance is warranted since an erroneous referral to a psychiatric facility may be embarrasing, costly, and delay proper diagnosis perhaps even to the point of permanent injury, disability, or premature death of a patient.

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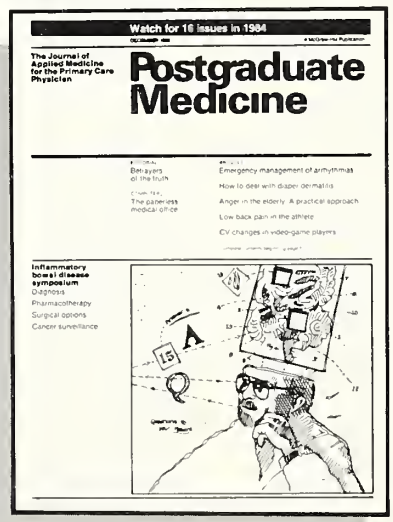
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My final presidential message is that the opportunity to visit each district is the most pleasant assignment of the state president. I am grateful to the members and their guests present at those meetings, who shared their knowledge and views with me, and for the hospitality extended to me by all districts.

My special "thank you" to our always faithful executive staff, who find ways to make things work out. Each deserves to be named. For now I will mention only Jeri Spars for patiently insisting that the page be prepared by deadline.

Dr. Howard Saylor of Huron is your president-elect. He will take over for the next edition. Many of you know Howard and his lovely wife, Mary Ann. If not, be certain to introduce yourselves during the annual meeting in Rapid City. I look forward to seeing you there.

Joseph N. Hamm, M. D., President
South Dakota State Medical Association

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Our SDSMA Auxiliary has enjoyed another challenging and successful year thanks to the enthusiastic and cooperative efforts of each auxiliary member, my State Board, our spouses, the South Dakota State Medical Association and the Association staff. It has been an honor to serve as auxiliary president.

Dr. Joe Hamm, SDSMA president, has been delightful to work with as well as being most supportive of the auxiliary. We are truly grateful for increased cooperation between our Medical Association and Auxiliary.

As president this year I was privileged to join auxiliaries around the state at district and board meetings, an AMA-ERF fundraiser and a Nurse's Tea. I became aware of the many members who are involved in various community projects to improve the quality of health. The hospitality extended to me was sincerely appreciated. I thank you all for it.

Our dedicated and hard-working committee chairmen have prepared the following interim update of activities for your information.

Judy Thompson, Members-at-Large: eight members have been retained/recruited. An increase over last year. **Karen Pekas,** Health Projects: researched and provided resources and materials to district auxiliaries focusing on the area of child abuse and neglect.

Mary Cosand, Legislation: represents auxiliary on Medical Association Commission on Legislation and Governmental Relations. Coordinated "Day at the Legislature" and dinner with legislators. With districts assistance we participated in a Legislative Alert which resulted in passage of HB 1086 in favor of Child Passenger Restraints.

Carolyn Nelson, Treasurer, and **Mollie O. Krafka,** Membership: as of March report a total membership of 389, state and national combined. This includes three male spouses. **Mary Barrett,** AMA-ERF: contributions to AMA-ERF and Medical Student Assistance Fund total $16,000 to date. **Karen Fletcher** and **Jane Solberg** continue to urge Resident Physician/Medical Student Spouses to join auxiliary.

I am proud to announce that Ila Lushbough, 1981-82 SDSMA Auxiliary president, has been nominated as an AMA Auxiliary National Director for our North Central Region. Ila was a National AMA-ERF committee member in 1983-84. In addition, I have been appointed to the AMA Auxiliary Legislation Committee for 1984-85.

Auxiliary members continue to work together to place our strength behind our physician spouses in their efforts regarding good medical legislation. We do urge all of you to join SoDaPAC in this important election year. This is an excellent opportunity for us to increase our participation in the political process.

See you at the annual meeting in Rapid City, June 7-10.

Marie Hovland

Marie Hovland, President
South Dakota State Medical Association Auxiliary

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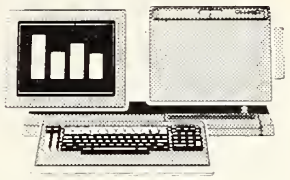

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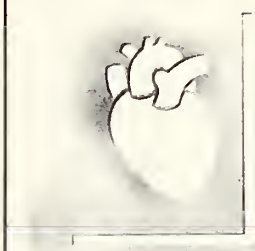

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Twenty Year Old Caucasian Male with Elevated Hematocrit and Right Lower Pole Renal Mass

Hoadley M. Harris, M.D.*
Richard A. Jaqua, M.D.†
Roger Nutt, M.D.‡
Discussers

John F. Barlow, M.D.§
Editor

Case #988 688

This 22 year old Caucasian male was admitted to Sioux Valley Hospital for evaluation of an elevated hematocrit and a right lower pole renal mass.

The patient had had bilateral testicular pain especially at night for two years, associated with painful erections. Ten months previously he had developed right flank and lower abdominal aching which was poorly localized and deep but with no definite radiation to the groin. There was no relationship to meals, or associated gastrointestinal or urinary symptoms. There was no hematuria. He was a nonsmoker. White blood count was 1.9 × 10^9/L, red blood count 8.02 × 10^{12}/L, hemoglobin 21 gm/dl, hematocrit 65 Vol/dl, mean corpuscular volume 81 fl, mean corpuscular hemoglobin 26 pg, mean corpuscular hemoglobin concentration 32%, red cell distribution width 14.7, mean platelet volume 8.5, platelet count 233 × 10^9/L, reticulocyte count 0.9%. A differential count showed 77% neutrophils, 1% neutrophilic bands, 19% lymphocytes, 2% monocytes, 1% eosinophils with 1 + anisocytosis, microcytosis, and poikilocytosis of the red cells on smear. A bone marrow examination showed erythroid hyperplasia. Arterial blood gas values, vitamin B-12 level, carboxyhemoglobin and hemoglobin electrophoresis were within normal limits. An erythropoietin level showed borderline elevation. A leukocyte alkaline phosphatase was depressed at 8. The patient was treated with multiple phlebotomies. The hematocrit dropped and the abdominal pain disappeared. The hematocrit again rose into the high 60's and a repeat erythropoietin level was markedly elevated. An intravenous pyelogram and computer tomographic scan (CT) showed a large, solitary, smooth cystic structure in the lower pole of the right kidney which was diagnosed as a possible simple cyst. He was referred for renal surgery. A hematology consult felt that the patient had secondary erythrocytosis.

A review of past medical history showed the patient had no other major symptoms. He had been evaluated at age 14 after an appendectomy because of short stature at which time he was 139 cm (4′ 1.5″) and 30 kgs (65 lbs). However, tests of the thyroid, adrenal and pituitary function were within normal limits. Additional tests included 12-panel chemistry which was normal except for an elevated alkaline phosphatase consistent with the patient's age. Skull and bone x-rays for age were unremarkable. Urinalysis was within normal limits and hematocrit was 41 Vol/dl with no other abnormalities on the complete blood count.

PHYSICAL EXAMINATION: Height 175 cm (5′ 2.5″); weight 48 kgs (106 lbs); pulse 80/min and regular; respirations 18/min and regular; blood pressure 120 systolic and 76 diastolic. The patient was alert, cooperative and in no distress. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The heart was not enlarged and had a normal rhythm with no murmurs or extra sounds. The abdomen was soft, flat and without tenderness, palpable organs or masses. The testes were descended. Rectal examination showed a smooth, nontender, small prostate. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis — yellow, clear; specific gravity 1.033; pH 6.5; negative for protein, glucose, ketones, bilirubin and hemoglobin; sediment negative. Hemoglobin 16.8 gm/dl, hematocrit 53 Vol/dl, mean corpuscular volume 76 fl, mean corpuscular hemoglobin 25 pg, mean corpuscular hemoglobin concentration 32%, total white count 7.2

* Resident in Family and Community Medicine, program affiliated with USD School of Medicine, Sioux Falls, SD.
† Professor and Chairman, Department of Laboratory Medicine, USD School of Medicine; Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD.
‡ Specialist in Internal Medicine; Clinical Faculty of Medicine, USD School of Medicine, Yankton, SD.
§ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

10^9/L with 48% segmented neutrophils, 45% lymphocytes, 2% eosinophils and 4% monocytes. The platelets were normal in morphology and the red cells showed slight anisocytosis and poikilocytosis on smear. The platelet count was 261 × 10^9/L. Lactic dehydrogenase (LD), alkaline phosphatase, asparate aminotransferase (AST, SGOT), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. An erythropoietin level was within normal limits. A chest film showed minimal calcific changes. An ultrasound-guided percutaneous puncture of the renal mass was attempted but no fluid could be aspirated. Internal echoes were seen within the cystic mass and an arteriogram was suggested. A right transfemoral selective renal arteriogram showed an avascular mass in the lower pole of the right kidney. An operation was performed.

DR. HARRIS: The signs and symptoms of renal neoplasms include hematuria, pain, palpable mass, hypertension, and manifestations of paraneoplastic syndromes.

Hematuria in renal neoplasms is more common in malignant than benign lesions and it may be gross or microscopic. Hematuria can also be seen in benign prostatic hyperplasia due to dilated bladder neck veins as well as in a variety of conditions involving the entire urinary tract.

Renal capsular distention is believed to be the cause of pain in the costovertebral angle region associated with renal neoplasms. Pain can also be caused by ureteral colic secondary to hyperperistalsis of the pelvis and ureter due to the passage of blood clots or fragments of tumor. Testicular pain is usually absent unless testicular torsion, bleeding, or infection are present. The painful erections in this case may be explained by erythrocytosis and the accompanying abnormal blood flow.

Palpable masses in the kidney include not only neoplasms but simple renal cysts, polycystic kidneys, and hydronephrosis. Pancreatic cysts or occasionaly splenomegaly can give palpable masses in the renal area.

An elevated blood pressure may be seen in up to 50% of the cases with Wilms' tumor but is rarely seen with renal cell carcinoma. Juxtaglomerular cell adenoma is a very rare renal neoplasm which secretes renin and produces hypertension in nearly all cases.

Paraneoplastic syndromes associated with renal tumors fall into four distinct areas. The first includes systemic manifestations manifested by fever seen in about 20% of renal cell carcinoma cases and secondary amyloidosis which can lead to renal failure and hepatosplenomegaly. The second encompasses hematologic manifestations. These include anemia seen in 35% of the renal cell carcinomas and erythrocytosis seen in 4% of renal tumors. Benign as well as malignant conditions of the kidney can be associated with an elevated hematocrit. In addition, an elevated erythrocyte sedimentation rate may be present. Thirdly, the gastrointestinal syndrome may present with abnormal liver function tests in the absence of hepatic metastases including abnormal prothrombin time, bilirubin, and alkaline phosphatase values which revert to normal after nephrectomy. This syndrome has been reported in up to 10-15% of the cases in some series. Lastly, endocrine manifestations have been seen. These include hypercalcemia secondary to metastatic bone disease or secondary to excretion of parathyroid hormone-like or prostaglandin-like substances from the kidney in the absence of metastases. Rarely ectopic secretion of adrenocorticotropin (ACTH) can produce Cushing's syndrome. Even rarer is gynecomastia secondary to secretion of gonadotropins by renal tumors.

I will now discuss some of the more common renal tumors. The only malignancy which tends to metastasize to the kidney is a neoplasm of the other kidney. Therefore, when we talk about renal neoplasms we are usually discussing primary tumors. Among the benign tumors is a cortical adenoma, a lesion derived from the renal tubular epithelium, which is seen in 7-22% of autopsies and is usually located in the cortex. By definition, it is less than 3 cm in size and it is composed of cells similar to those seen in renal cell carcinoma. Some feel this is simply a small renal cell carcinoma.

An oxyphil adenoma or oncocytoma of the kidney is a benign lesion which can reach large size and has a characteristic cell with abundant red cytoplasm filled with mitochrondria on electron microscopy.

The fibroma is composed of well differentiated fibroblasts and is a small lesion usually found in the renal pyramids at autopsy. It has no malignant potential.

The so-called renal hamartoma or angiomyolipoma is commonly seen in tuberous sclerosis, however, it may be seen in the absence of that disease and is composed of vessels, smooth muscle and fat. It is a benign lesion but may undergo spontaneous rupture or hemorrhage and cause pain and anemia. The lesions can often be diagnosed by angiography which shows multiple small aneurysms.

Hemangiomas may be composed of blood vessels or lymph vessels and are benign congenital lesions which usually occur in the medulla of the kidney. Their most common site is the liver.

Malignant tumors — Renal cell carcinoma (Grawitz tumor or hypernephroma) was described in 1883 and was named hypernephroma because it appeared similar on gross appearance to the adrenal cortex. These lesions constitute 85% of the primary malignant renal tumors. It is totally unpredictable. The primary may be silent and the presenting symptoms may be due to metastases to the brain, bone or lung. These metastases may be single and some have

suggested that if they are removed along with the primary, reasonable survival can be expected in some cases. The renal primary may grow to large size and be silent without metastases. On the other hand, growth may be explosive. Therefore, prediction of survival is very difficult. The microscopic morphology of these tumors is also highly variable. As mentioned, cortical lesions below 3 cm in diameter with typical well differentiated histology are classified as cortical adenomas by some authors. Renal cell carcinomas usually appear after the fourth decade, and often in the upper poles of the kidney as solitary lesions. They are spherical masses 3-15 cm in diameter and are bright yellow or gray. They show pseudoencapsulation but are not truly encapsulated. Over 50% will invade the renal vein or grow in a column of tumor into the inferior vena cava. Invasion of these vascular structures as well as invasion of the renal capsule and surrounding perinephric region are considered poor prognostic features. The tumor characteristically is very vascular, a feature helpful in angiographic diagnosis. There are a variety of histologic patterns including papillary, tubular, glandular, and undifferentiated forms such as sarcomatoid varieties. The clear cell carcinoma variant is the most common and has many of the ultrastructural features of tubular epithelium. This clear cell pattern can often help the pathologist in recognizing the primary when a metastasis is removed from a distant organ. Since there is abundant lipid in the cells, the presence of fat in aspirates of renal masses may be sign of malignancy.

I will now return to some of the classic clinical aspects. Hematuria is seen in 60-90% of the cases but pain and a palpable abdominal mass are usually late symptoms. It should be pointed out that the classic triad of hematuria, pain and palpable abdominal mass is only seen in 9% of the cases of renal cell carcinoma. Hematuria in conjunction with the paraneoplastic syndromes, accounts for most presentations of renal cell carcinoma. A high index of suspicion followed by radiologic diagnosis in renal cell carcinoma is a must because of the wide variety of symptoms. These tumors metastasize hematogenously to the lungs (50%) bones (33%) and to the opposite kidney (15%) as well as to lymph nodes, liver, brain and adrenals. Because the tumor exhibits bizarre growth patterns and may be slow growing and silent or grow in explosive nature as well as having a variety of histologic patterns, it is a most confusing tumor.

Treatment is also perplexing. A renal neoplasm thought to be cured by surgery may develop metastasis 10-20 years later. The overall 5 year survival is 35%. This rises to 50% if it is confined to the kidney (stage I). The prognosis drops to the 10% range if the renal vein or perinephric fat are involved. Chemotherapy and radiation have been only slightly successful in treating this neoplasm. The major hope of cure is extirpative surgery at an early stage.

The second most common malignant tumor of the kidney is the Wilms' tumor (embryoma or nephroblastoma). This tumor is second only to neuroblastoma as a solid malignant neoplasm in viscera in children. Sixty-five percent of cases occur in children under 3 years of age. The tumor contains a variety of cellular tissues so that some believe it is a form of teratoma. The tumor is usually a large expansile unilateral mass which can produce abdominal distention with a palpable mass. In fact, most children present with an abdominal mass while hematuria and abdominal pain are less common. The tumor may cause intestinal obstruction and pulmonary metastases are not uncommon at the time of tumor diagnosis. Fortunately, with a combination of chemotherapy, radiation and surgery, a two year survival of over 90% even with metastases to the lungs has been achieved. When metastases occur to other organs such as the bone, brain and liver, the survival is less.

What are some of the diagnostic modalities used for diagnosis of renal tumors? In the abdominal plain film an enlarged kidney with bulging contour and obliteration of the psoas shadow are indications of renal neoplasm. The psoas shadow may be seen through a cyst but not a solid tumor.

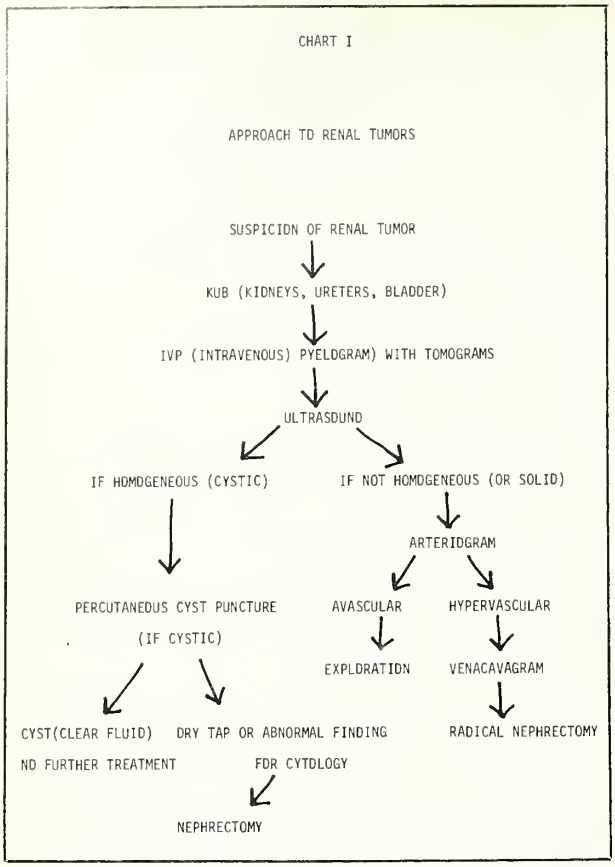
In the intravenous pyelogram, a filling defect or distortion of the calyceal pattern may occur. Very rarely a renal cell carcinoma may compress the ureter and produce secondary hydronephrosis. If renal vein involvement occurs, there may not be excretion of radiocontrast material by the kidney. This is a bad prognostic sign. Tomograms of the kidney may show the tumor to better advantage.

The sonogram is very helpful in diagnosis of renal tumors because it can distinguish cysts from solid neoplasms in 95% of the cases. If there is any question, a percutaneous needle puncture may be obtained. If clear fluid is obtained, the lesion is a cyst. If bloody fluid is obtained, a tumor may be present and cytologic examination of the fluid should be performed. Biopsy of renal neoplasms can also be carried out under sonographic guidance.

There is some question whether the computer tomogram is of any additional help in differentiating cyst from solid tumor in the kidney.

However, angiography is of considerable value because of the characteristic neovascularity in renal cell carcinoma. Pooling may also occur in the tumor but not in the cyst.

Finally, I would like to outline an overall approach to the diagnosis of renal tumors (Chart I). First, one must be very suspicious of a renal tumor.



A simple x-ray of the kidneys, ureter and bladder (KUB) may show a mass or an enlarged kidney with an irregular outline. An intravenous pyelogram (IVP) with tomograms is the next step. A sonogram to distinguish between a cystic or solid mass can then be performed.

Cystic lesions can be aspirated and cytology performed if fluid is bloody. No further treatment is usually necessary although the wall can be destroyed by sclerosing solutions. If the tap is dry or abnormal cells are present on cytologic examination, nephrectomy is indicated. If the lesion is not homogeneous by sonogram, an angiogram is necessary. If a typical hypervascular pattern is seen, a radical nephrectomy perhaps preceded by an inferior venacavagram is necessary. Exploration is also indicated for avascular neoplasms as in this case.

The tumor we had today was in the wrong age group for a renal cell carcinoma and was avascular on the angiogram. I think from the studies I can safely say it was a neoplasm and not a cyst. Other than that, I can not be more specific.

Dr. Harris's Diagnosis
Neoplasm of Kidney

DR. BARLOW. Thank you for your excellent discussion. I agree this is a most unusual neoplasm and would be impossible to diagnose without pathologic examination.

Submitted was a 140 gm 11 × 5.5 × 3.5 cm right kidney. In the lower pole was a 4 cm in greatest dimension smooth tan homogeneous well demarcated mass which bulged from the surface but did not invade the perirenal fat (Fig. 1). Microscopically the lesion was well demarcated and in some areas a pseudocapsule was seen. There was no invasion of the surrounding parenchyma. Most of the tumor was composed of masses of tubules lined by cells with scanty cytoplasm but uniform round to oval nuclei without hyperchromatism. Mitoses were rare. In other areas a marked papillary arrangement with psammoma bodies was present (Fig. 2). Structures consisted with glomerulotubular structures but probably papillae were seen in solid areas of the tumor. Necrosis, macrophage infiltration or collections of foam cells were not seen.

Figure 1
Gross picture of cut section of solid well circumscribed renal tumor.

One thought was that this might represent a Wilms' tumor in an adult. However, the frank papillary areas and psammoma bodies made this unlikely. In addition, the tumor was sent to Dr. George Fairow of the Mayo Clinic, Department of Surgical Pathology and he felt the lesion was a papillary carcinoma.

I think it is of interest that 167 cases of adult Wilms' tumor have been reported in adults. Some of these may represent sarcomatoid variants of renal carcinoma and some authors may have confused papillary arrangements with glomerulotubular structures characteristic of Wilms' tumor (Fig. 3).

DR. JAQUA: When the light microscopy of this tumor was presented at our noon surgical conference there were two questions which I felt ultrastructure might help answer. The first would be to look for

mesenchymal or glomerular type differentiation to help differentiate Wilms' tumor from a papillary adenocarcinoma, and secondly to see if we could demonstrate any ultrastructural evidence for secretory activity in the tumor to correlate with the elevated erythropoietin levels.

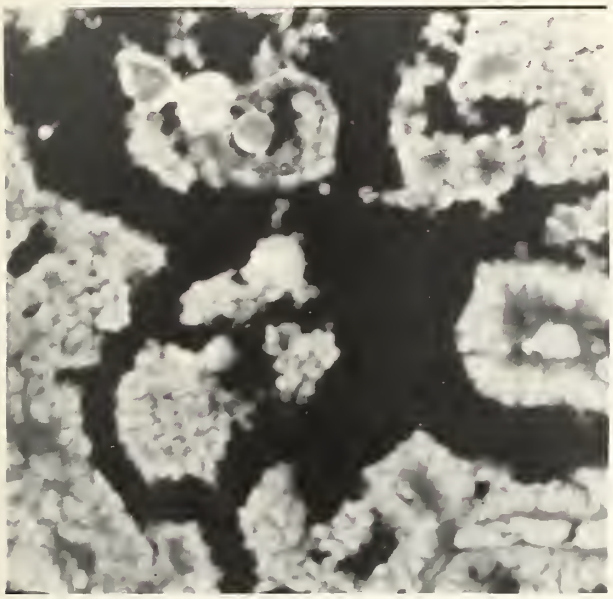


Figure 2
Papillary features with psammoma bodies (upper center).

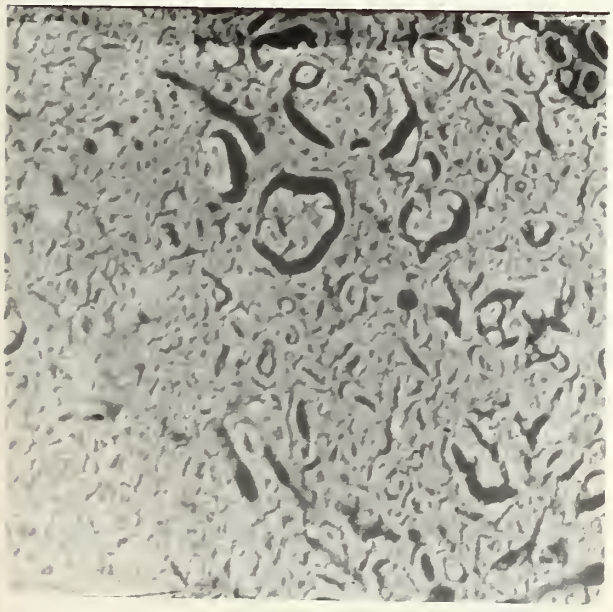


Figure 3
Pseudoglomeruloid structures suggestive of Wilms' tumor.

We examined numerous blocks from this tumor by both thick and thin section morphology and were unable to demonstrate any mesenchymal or glomerular type differentiation. The tumor was nearly entirely made up of tubular-like structures with well developed basal lamina, a small, often collapsed lumen and with polarized columnar cells exhibiting well developed junctional complexes at their apices along with true microvilli and an occasional cilium extending into the lumen (Fig. 4). No secretory activity was identified in the epithelial cells or in stromal cells. Unlike renin, erythropoietin has not been localized to an ultrastructurally defined granule, and although studies are conflicting, the immunochemical studies which have been done seem to point to the glomerulus as the potential renal source. Since this tumor tissue was not assayed for renin, the possibilities of tumor compression of normal renal parenchyma and secondary prostaglandin stimulation of erythropoietin must also be considered in this case. I believe the ultrastructure confirms the renal epithelial origin of this neoplasm.

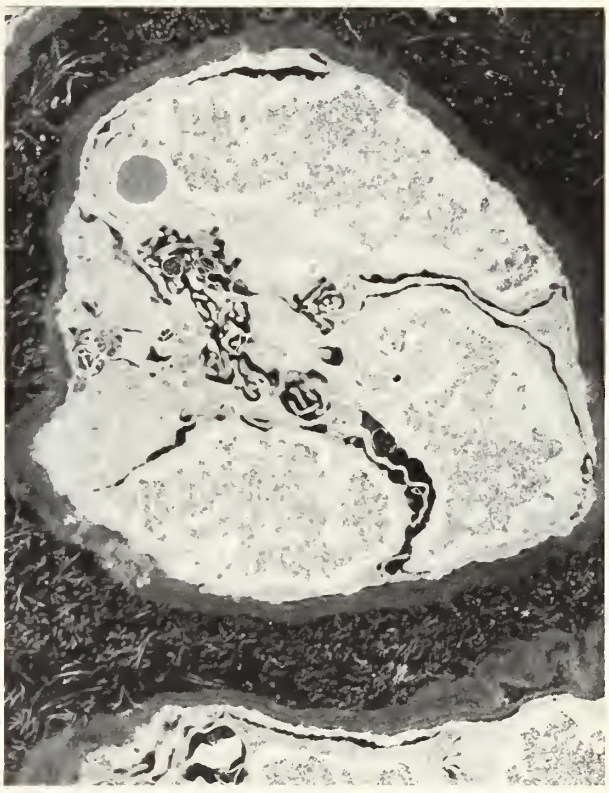


Figure 4
Electron micrograph showing cells in a tubular arrangement favoring renal tubular origin of this tumor.

DR. BARLOW: Papillary carcinomas are rare tumors. One series reported 34 cases culled from 224 renal cell carcinomas. The majority of papillary carcinomas (85%) were confined to the kidney (stage I) compared to only half of the nonpapillary varieties. Papillary carcinomas had a better survival rate than other renal cell carcinomas even though massive necrosis, a poor prognostic feature of nonpapillary carcinomas, was frequently seen in the papillary renal neoplasms. Psammoma bodies and

calcification identified on x-ray was common. An interesting finding was atypical proliferation of the collecting tubules near the tumor suggesting an origin from these structures. This was not noted in this case. It must be stressed, however, that these tumors can invade the renal vein and produce multiple metastases, but lesions which did so did not show features considered to be associated with a good prognosis — encapsulation, necrosis or macrophage infiltration. The lesion today can probably be considered encapsulated but did not show macrophage infiltration or necrosis. The fact that the lesion was confined to the kidney (stage I) would indicate a good prognosis.

Papillary tumors characteristically are hypovascular or avascular on arteriography, a finding nicely demonstrated by this case (Fig. 5).

Figure 5
Arteriogram of kidney with absent vascularity in lower pole of kidney of this case typical of papillary carcinoma.

Although papillary carcinomas of the kidney have been associated with hypertension and hypercalcemia, I did not see mention of a case with erythropoietin production and secondary erythrocytosis. Secondary erythrocytosis has been described in over 350 neoplasms. Over half or 179 have been from the kidney. One hundred twenty of these were hypernephromas. Wilms' tumor, sarcomas, and renal cysts have also been known to secrete erythropoietin. However, only 1-10% of all renal cell carcinomas cause secondary erythrocytosis and only 1% of cases of erythrocytosis are due to renal cell carcinoma. There was a study showing that 63% of cases of renal cell carcinoma secrete erythropoietin but this does not usually result in an elevated hematocrit. This latter finding has been associated with an improved prognosis.

FINAL ANATOMIC DIAGNOSIS
PAPILLARY CARCINOMA OF THE RIGHT KIDNEY

*DR. KEITH A. ANDERSON: Would you clarify that point on the presence of fat in cytologic preparations from aspiration of renal lesions?

DR. HARRIS: As I understand it, there seems to be a high correlation between the presence of high lipid levels in fluid aspirate and the presence of malignancy.

DR. BARLOW: I would like to introduce Dr. Roger Nutt, the physician who referred this case to Dr. John Sall who subsequently operated on this patient.

DR. NUTT: I can provide some followup. The patient is doing extremely well and now has an hematocrit of 42% several months after surgery.

†DR. TOM GAECKLE: What do you feel is the mechanism for erythropoietin secretion in this case? Isn't it true that erythropoietin secretion occurs less commonly in cysts than tumors of the kidney?

DR. BARLOW: I am sure it depends in what series you read, but if one were to take autopsy series, I am sure erythropoietin secretion by renal cysts would be considerably less than 1%.

DR. JAQUA: I think one has to realize that some feel that it is the compression of the surrounding kidney which is responsible for erythropoietin or renin secretion by the kidney in the case of any expansile condition such as renal cyst or renal tumor. This may be mediated by prostaglandin elevation which in turn stimulates increased erythropoietin levels. Juxtaglomerular tumors have been described that actually secrete prostaglandins and elevate erythropoietin in this fashion.

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* Resident in Pathology, Sioux Valley Hospital and Veteran's Administration Hospital, Sioux Falls, Program Affiliated with School of Medicine, University of South Dakota, Sioux Falls, SD.

† Specialist in Internal Medicine, Sioux Valley Hospital, Clinical Faculty of USD School of Medicine, Sioux Falls, SD.

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Advances in Hepatic, Biliary and Pancreatic Surgery, Willey Hall, U. of Minn., Minneapolis, MN, June 13-16. Fee: $400. 26 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Daniel C. Carrow Education Day (Pediatrics), Battenfeld Aud., U. of Kans. Med. Ctr., Kansas City, KS, June 20. AMA Category I credits. Contact: Dept. of Peds., U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-6323.

Clinical Hypnosis, Earle Brown Ctr., U. of Minn., St. Paul, MN, June 22-23. Fee: $275. 13.5 hrs. AMA Category I, AAFP & APA credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Human Aging: Senile Dementia of the Alzheimer's Type, Willey Hall, U. of Minn., Minneapolis, MN, June 27-28. Fee: $75. 13 hrs. AMA Category I & AAFP credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Real Time Ultrasound in Obstetrics and Gynecology, Malcolm Moos Hlth. Sci. Tower, U. of Minn., Minneapolis, MN, June 27-29. Fee: $475. 19 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

July

Requested Topics for Clinicians, Marriott Hotel, New Orleans, LA, July 13-15. Fee: $225. 11 hrs. AMA Category I credits. Contact: Dennis M. Occhipinti, M.D., 5640 Read Blvd., Suite 400, New Orleans, LA 70127.

Pediatric Orthopaedic Surgery, Hyatt Regency, Minneapolis, MN, July 30-Aug. 1. Fee: $350. 18 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

August

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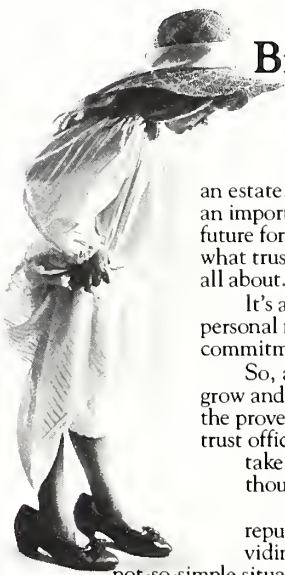

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SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 6 June 1984 (ISSN 0038-3317)

Official Publication of the
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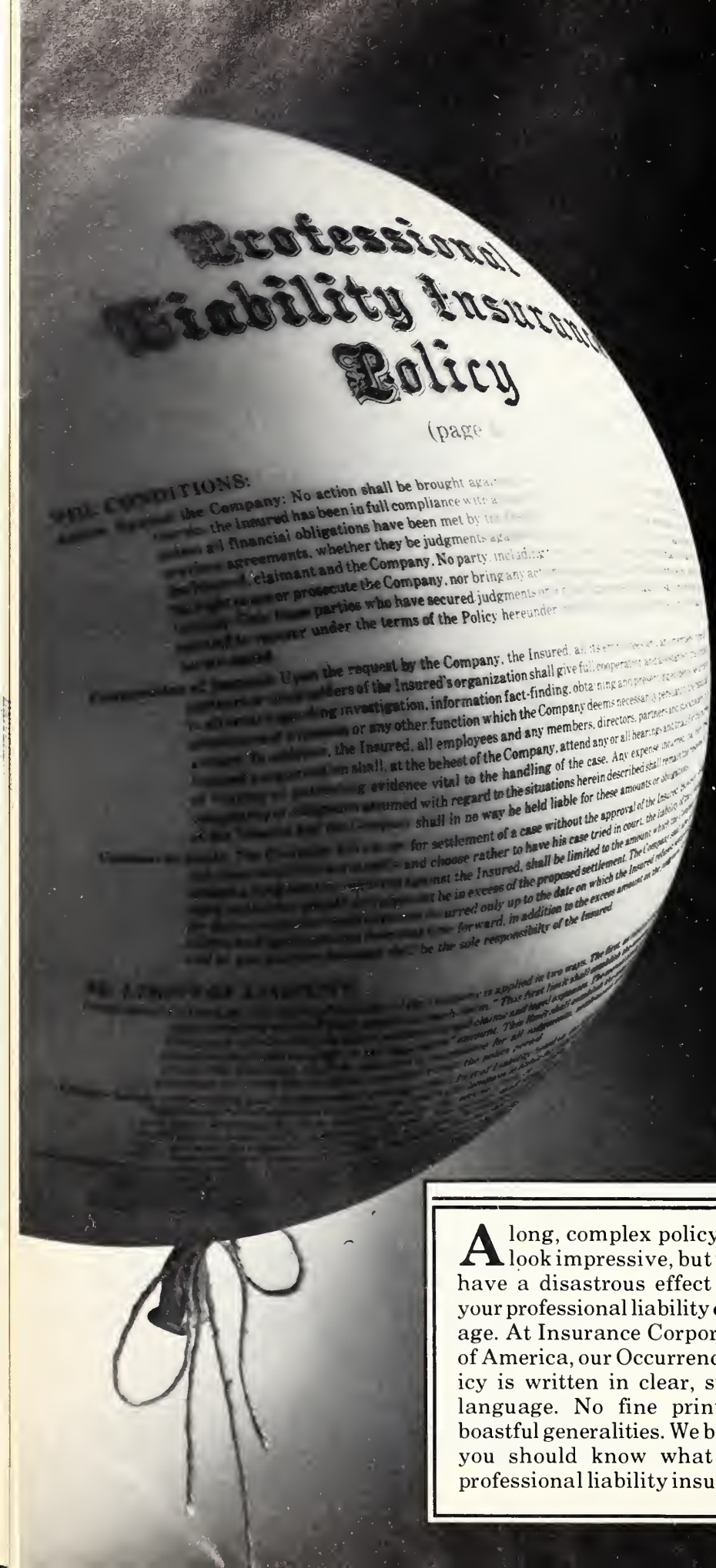
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Tularemia With Unusual Laboratory Characteristics in South Dakota Children

Bert W. Larson, M.D.*
Helen J. Jacobson, M.T., A.S.C.P., S.M.†

ABSTRACT

Clinical findings associated with three recent isolates of Francisella tularensis from South Dakota children are presented. Observations are made on clinical and laboratory features and on antibiotic susceptibility. Unusual growth characteristics of the isolates are described, and their relevance to laboratory diagnosis and safety is discussed.

INTRODUCTION

Tularemia has been infrequently identified in South Dakota, with only eleven cases reported between 1970 and 1979.[1] In our laboratory at Sioux Valley Hospital in Sioux Falls, South Dakota, **Francisella tularensis** (formerly **Pasteurella tularensis**) was identified from three specimens between May and July of 1983. The patients were children between the ages of three and ten years who had ulceroglandular tularemia, apparently arthropod-borne. Each isolate showed unusual growth characteristics, appearing on media not widely thought to support the organism. Despite delayed streptomycin therapy, each child ultimately responded nicely to the drug.

CASE REPORTS

Case 1

A six year old female was bitten on the left ankle by an arthropod which she felt was a spider on approximately 4-13-83. Two days later she developed a high fever and ipsilateral inguinal adenopathy and was treated as an out-patient with oral penicillin and subsequently with an oral cephalosporin for nine days. She was then treated in her local hospital with intravenous cefamandole prior to her transfer to Sacred Heart Hospital in Yankton, South Dakota on 4-26-83.

At Yankton, the child was found to have an undulating fever to 103 degrees F, an 8 mm ulcer on the left medial malleolar area with surrounding erythema, and a tender dome-shaped non-fluctuant mass in the left inguinal region. A white blood count early in the course was 17.3 $\times$ 10^9/L with a differential of 57% segmented neutrophils, 6% neutrophilic band forms, 35% lymphocytes, 1% monocytes, and 1% basophils. A lymph node biopsy was obtained on 4-28-83 from the left groin which showed foci of acute necrosis with surrounding lympho-histiocytic proliferation (Fig. 1). A portion of the node was retained for culture. A pediatric consultant suggested the possibility of tularemia and on 4-28-83 streptomycin therapy was begun in a dose of 250 mg intramuscularly every 12 hours. The child was afebrile within 36 hours and was doing well when discharged on 5-2-83 to continue therapy in her home town on an outpatient basis.

On 5-10-83 our laboratory received a chocolate agar plate from Sacred Heart Hospital. It had been streaked from a subculture of a gram negative bacillus which their laboratory had grown on enriched chocolate agar from tissue obtained at lymph node biopsy. The fresh inoculum was incubated in a 5-10% CO_2 containing environment in our laboratory, and in two days a small gram negative rod appeared.

* Resident in Pathology, University of South Dakota Affiliated Hospitals, Sioux Falls, SD.
† Chief Technologist, Microbiology Laboratory, Sioux Valley Hospital, Sioux Falls, SD.

This was inoculated on additional media and within 48 hours the organism was growing well on enriched chocolate and 5% sheep blood agar, though not on MacConkey or EMB agar. It was identified as **Francisella tularensis** using a specific agglutinin and this was later confirmed by the Centers for Disease Control. Results of febrile agglutinin studies performed on 4-28-83 were subsequently received which showed a positive tularemia titer of 1:320.

Case 2

A three year old male became ill on 7-2-83 and was hospitalized in a rural South Dakota hospital with suspected pneumonitis. His fever was 104 degrees F with an associated white blood count of 24.5 $\times$ 10^9/L, the differential showing 81% segmented neutrophils, 3% neutrophilic band forms, and 16% lymphocytes. A small right middle lobe infiltrate was apparent on chest x-ray. Additional history revealed that he had been bitten by a tick on the left ear two weeks prior to admission and had subsequently developed impetigo in this site. A swelling which was present in the left cervical region enlarged during this hospitalization. Treatment at the local hospital included incision and drainage of the left cervical area and antibiotic therapy consisting of intravenous nafcillin.

The child was transferred to Sioux Valley Hospital on 7-12-83 due to persistent erythema and swelling in the left neck, extending from the postauricular region to the submandibular area. He showed undulating fevers to 104 degrees F. Initial white blood count was 15.8 $\times$ 10^9/L with a differential of 49% segmented neutrophils, 11% neutrophilic band forms, 33% lymphocytes, 4% monocytes, and 3% eosinophils. A chest x-ray was negative. On 7-13-83 repeat incision and drainage was performed and cultures of the material were made. Sections of tissue from the area showed acute and chronic inflammation and necrosis. Intravenous cefamandole was then given for three days without benefit. A pediatric consultant suggested that the ailment was probably tularemia, and streptomycin therapy was begun at a dose of 250 mg intramuscularly every 12 hours. The patient was afebrile within 24 hours and was discharged much improved to his local hospital to continue streptomycin therapy.

Cultures from material obtained at incision and drainage produced growth of a gram negative rod on enriched chocolate and 5% sheep blood agar which was identified as **Francisella tularensis** using specific agglutinating antiserum. Confirmation of identity was obtained from the Centers for Disease Control. A febrile agglutinin study performed on 7-16-83 was positive for tularemia in a titer of 1:320.

Case 3

A ten year old Sioux Falls boy was seen by his physician on 7-18-83 with a three day illness consisting of a gradually increasing temperature to 105 degrees F. When seen in the doctor's office he had a white blood count of 15.8 $\times$ 10^9/L, mild proteinuria and ketonuria, and emesis, as well as a mild gait imbalance. There was a history of a cat scratch on the left arm one month prior to admission with subsequent healing and scar formation. The patient was admitted to Sioux Valley Hospital where further examination revealed right axillary adenopathy and numerous insect bites on the extremities. There was an area of inflammation on the right index finger. The white blood count was 9.2 $\times$ 10^9/L with a differential of 50% segmented neutrophils, 33% neutrophilic band forms, 16% lymphocytes, and 1% monocytes. The lesion on the right index finger became vesicular, and a culture from the vesicle was obtained on 7-22-83. Meanwhile, treatment had been started with erythromycin and the patient was gradually defervescing. The cutaneous lesion was unimproved, however.

Within 48 hours, growth of a gram negative rod was noted on enriched chocolate agar, though not on sheep blood agar or other media. The culture was identified as **Francisella tularensis** using specific antiserum and was referred to the Centers for Disease Control and confirmed. Therapy was begun with streptomycin 500 mg intramuscularly every 12 hours, resulting in rapid improvement in the condition of the right index finger and axilla. The child remained afebrile, continued to improve and was discharged on 8-1-83.

These cases, all occurring in children in the southeastern quadrant of South Dakota, had several features in common:

1. **Each case had evidence of an arthropod vector.**
2. **Each child had ulceroglandular disease (one patient, Case 2, also had evidence of pneumonitis early in his course).**
3. **Prior to diagnosis each child received one or more antibiotics to which Francisella tularensis is considered resistant.**
4. **Each case resulted in culture of the organism on enriched chocolate agar, and in two cases, sheep blood agar also supported the organism.**

These features are the basis for the following review.

MODE OF TRANSMISSION

The classic source of tularemia is the rabbit. It has been written (despite earlier contrary reports[2, 3]) that the disease occurs almost entirely in the winter

months during the rabbit hunting season.[4] The children presented here contracted tularemia in the warmer months without known contact with rabbits or other mammals known to carry the disease such as squirrels or muskrats. Each child, however, was exposed to an arthropod vector.

The tick (Case 2) is a well recognized vector. Older literature suggests the association of tick bites and tularemia.[2, 3] Transmission has been linked to tick bites in over one-half of pediatric cases[3] and to a similar proportion of head and neck cases.[5] The tick is the most common vector in the central United States[1] and is by far the most common vector west of the Mississippi River,[6] where arthropods are often associated with the disease.[7] In the East, tularemia is second only to Rocky Mountain fever among tick-borne disorders.[6]

The deer fly is also a recognized vector.[7] An ailment known in Utah as "deer fly fever" early in this century was most probably tularemia.[2] Mosquitoes may transmit tularemia.[7] Case 3 in this series had numerous apparent mosquito bites without other obvious exposure, suggesting the possibility of mosquito-borne illness. Of incidental interest is that this patient had a history of a cat scratch, though it occurred a month prior to onset (the incubation period of tularemia ranges from one to fourteen days[5]) on the limb opposite the primary lesion. The domestic cat is an uncommon but documented source of infection.[8, 9] This usually results from a bite rather than a scratch and may reflect contamination of the mouth of the animal from its prey. Domestic pet exposure seems unlikely as a source of tularemia in the patients described here. A cutaneous lesion with local adenopathy following such exposure would more likely be due to **Pasteurella multocida** (a distantly related gram negative rod), or possibly cat scratch disease or toxoplasmosis.[10]

TYPE OF ILLNESS

Each patient presented with ulceroglandular tularemia, the most common form of the disease. Fever, a skin ulcer or vesicle, and adenopathy in a distribution draining the cutaneous lesion were present in each case. These are the common features of tularemia, and an associated history of arthropod exposure nearly secures the diagnosis. Delay in therapy may result from failure to consider tularemia since early manifestations (headache, fever, and chills) are common to other febrile ailments.[5] A careful search for a local lesion should therefore be made in all cases of regional lymphadenitis,[3] and a careful history, particularly for tick exposure, should be taken.[6] This is especially true since agglutination titers do not appear until week two of the illness[7] and may not be clinically significant until weeks later. Fatal cases have occurred in the absence of a positive agglutinin titer.[11]

One must consider the disease in patients with less common presentations as well. These are often associated with handling or ingestion of infected animals. Pharyngitis is a well documented form in pediatric patients, in whom it may be indistinguishable from diphtheritic, streptococcal, or mononuclear pharyngitis.[11] This has been said to be more common in younger children.[3] Pneumonitis may be seen in association with any form of the disease and may be acquired through the bloodstream or by direct inhalation of the organisms. It may complicate 21% to 40% of cases[6] and may resemble other pulmonary infections such as tuberculosis and legionellosis.[10] Pneumonic disease may be seen in urban centers,[12, 13] a reminder that the classic rural presentation need not be present. The typhoidal form, often seen in laboratory-acquired infections,[6, 14] is a generalized febrile ailment in which there may be no glandular enlargement to serve as a diagnostic clue. It may begin with a flu-like state.[14]

Figure 1
Lymph node biopsy from Case 1, showing a focus of necrosis with surrounding lympho-histiocytic proliferation.

Mortality in untreated cases of tularemia may be significant. It is variably reported as 5% to 30%[1] and 5% to 40%[10]; however, an older series from the pre-antibiotic era[2] showed a mortality of 4% in 123 varied cases. The disease is definitely less virulent than its relative, plague (which, though customarily considered a disease of the Southwest, is occasionally seen in our neighboring state, Wyoming[10]). In fatal cases, there is a striking resemblance to tuberculosis. Autopsy cases[2, 3] may show gray tubercles throughout the viscera, consisting of evolving areas of necrosis with a surrounding granulomatous reaction. A suggestion of this lesion is seen on sections of an inguinal lymph node biopsy from Case 1 of this report (Fig. 1). Although disseminated tularemia is

likely to occur today only in badly neglected cases, consideration of such cases should direct the differential diagnosis.

ANTIBIOTIC THERAPY

Each of these children was treated with therapy directed presumably against gram positive organisms. This is not surprising since cutaneous infections and lymphadenopathy in children not infrequently are caused by Streptococci or Staphylococci. In Case 1 penicillin and cephalosporins were given; Case 2 received intravenous nafcillin and cefamandole; in Case 3 initial therapy consisted of oral erythromycin. The tendency to use therapy of this type early in the course of tularemia is in no way unprecedented. Treatment with penicillin and a cephalosporin prior to diagnosis is seen in other series.[5, 15] Therapy with dicloxacillin, clindamycin, and ampicillin over a six day period prior to growth of the organism is described in a pediatric patient from the University of Minnesota Hospitals.[7] Thus, one must remember that whereas Streptococci and Staphylococci are admittedly common agents, they are also readily cultured, and failure to grow these organisms from the primary lesion or infected bubo is a signal that an uncommon organism may be present. Cephalosporins, although generally effective against Streptococci and Staphylococci, remain unsatisfactory or second-line drugs in many settings despite their wide use. Failure of response to penicillin in the absence of growth of the ubiquitous **Staphylococcus aureus** should signal restraint in their use in lymphadenitis.

Our isolates were characterized by in vitro resistance to ampicillin, carbenicillin, cephalothin, and trimethoprimsulfamethoxazole and sensitivity to aminoglycosides as well as tetracycline and chloramphenicol.

The latter agents have been used successfully in tularemia. Susceptibility to these drugs make them superficially attractive, particularly the less toxic tetracyclines in patients who have their secondary dentition. However, it must be remembered that these are bacteriostatic agents, and the immune system must be relied upon to fully irradicate the organism. Thus, relapse is a threat when these agents are used and has been described even after a seven to ten day course despite good initial response to these broad spectrum agents.[14]

Among the aminoglycoside antibiotics, streptomycin is the prototype and was first identified as the drug of choice for tularemia in 1948,[10] shortly after it became available. Whereas rapid development of resistance to streptomycin is considered common with other organisms such as Shigellae,[16] it is not described with Francisella. Therefore, it remains the drug of choice today in those who can tolerate it. Observance of vestibular and auditory function and appropriate lowering of the dose in renal patients is advised. Although most regimens utilize a seven to ten day course, using a dose varying from 15 to 40 mg/kg/day[5, 10, 16] in two daily intramuscular doses, none has been proven superior. It has been suggested that a shorter course may be acceptable.[10] Repeatedly emphasized in the old as well as recent literature[3, 7, 11] is the maxim that a trial of streptomycin is strongly indicated in suspected cases since confirmatory evidence of the diagnosis may have to await serologic data. Two of the patients in this series (Cases 1 and 2) received therapy with streptomycin prior to confirmation of the disease, and they responded appropriately. A brisk response to therapy, with defervescence in 48 hours[3] plus supportive history and physical findings, is strongly suggestive of tularemia.

The more contemporary aminoglycoside antibiotics may also have a place in the treatment of tularemia. Gentamicin has been investigated in adult patients,[15] and it is effective in vivo as it is in susceptibility testing. It is suggested that its use is particularly valuable for situations in which the diagnosis is not firm but is suspect and the patient's condition requires that more broad spectrum coverage be given than can be afforded by streptomycin alone. Amikacin and tobramycin are as yet unproven for use in tularemia, although an anecdotal case[17] suggests the efficacy of the former.

GROWTH AND LABORATORY CHARACTERISTICS

Finally, of interest in these cases is their behavior in the laboratory. It is said that routine hospital laboratories rarely recover **Francisella tularensis** because of its growth requirement for cysteine or cystine.[7] In fact, one of the screening criteria for identification of the organism is its sluggish growth on blood agar (which supported the organism in two of our cases) as contrasted with its ready recovery on glucose cysteine agar or cystine heart agar.[18] Furthermore, it has been widely stated that laboratories should not attempt to grow the organism[11, 15] since there is a documented risk of laboratory acquired tularemia, even in immunized individuals.[14]

A number of questions arise:

1. **Is it true that a special Francisella agar is required for growth, or do routinely available media sustain growth?**
2. **What is the hazard to laboratory personnel?**
3. **If a laboratory elects to attempt isolation of this organism or inadvertently isolates it on routine media, what precautions should be taken?**

4. **If one wishes to culture the organism, what are appropriate methods?**
5. **What are the alternatives to culture in making the diagnosis?**

These issues are discussed individually below.

1. Although highly cysteine or cystine enriched agars are accepted as preferred media for culture of Francisella, growth has been accomplished on other media, such as selective media for gonococci (including Thayer-Martin) and enriched chocolate agar.[18, 19] Our chocolate agar contains Bacto-Supplement VX (Difco Laboratories) enrichment supplement in addition to a GC medium base (Gibco Laboratories) and hemoglobin. This provides 259 mg of L-cysteine HCL and 11 MG of L-cystine per 1000 ml of distilled water in addition to other nutrients. It contrasts with 1 gm of cysteine or cystine per 1000 ml water present in glucose cysteine agar or cystine heart agar. Nevertheless, the cases described suggest that Francisella may appear with some reliability on modern enriched chocolate media. Thus, the organism may appear in culture even if the laboratory does not choose to grow it. Alternatively, enriched chocolate agar may be a suitable medium on which to inoculate a suspect specimen if the classic Francisella media are not immediately available.

Our experience in two of the cases (Cases 1 and 2) suggests that the organism may occasionally appear on sheep blood agar as well. Our sheep blood agar plates contain Vitamin K Hemin Solution (Gibco Laboratories) in addition to tryptic soy agar (Difco Laboratories) and 5% sheep red cells. Growth on this medium in the absence of added cysteine or cystine is generally considered uncharacteristic of Francisella, but it reaffirms the premise that it may be grown unintentionally in a routine microbiology laboratory.

2. The risk of acquiring tularemia in the laboratory is not small, particularly in individuals who work continuously with the organism.[14] The bacterium is said to be capable of penetrating intact skin,[1] and less than 50 organisms may constitute an infective dose by the intracutaneous or respiratory route.[6] The occurrence of the disease in the absence of a laboratory accident and its occurrence in individuals with a remote contact with the organism also suggests high infectivity.[1, 14] It can be assumed that the risk to laboratory personnel is real, though we are unaware of any cases having resulted from laboratory exposure to our isolates.

3. Appropriate isolation technique and reasonable laboratory precautions are suggested in the **Manual of Clinical Microbiology.**[19] It is recommended that laboratory workers use surgical gloves in handling the plates, employ a vented flow hood, and avoid aerosolization in working with Francisella. Vaccination has been used as a preventive measure in those who routinely work with the organism, but this has not been entirely successful in preventing infection.[14, 20] The severity of infection did not correlate with previous vaccination in one series,[14] while in another the milder ulceroglandular form was found to occur more frequently than the typhoidal form in previously vaccinated workers.[20] This was consistent with the characteristics of repeat infections in nonimmunized but previously infected hosts. The vaccine is apparently obtainable from the Center for Disease Control, but its utility in a routine laboratory setting is open to question.

4. Two of the present cases grew from infected surgical material, one from a bubo removed at surgery (Case 1) and one from material obtained by incision of affected cervical lymph nodes (Case 2). The final case was isolated from a primary vesicular cutaneous lesion. These types of infected materials would be the most frequent sources of culture specimens in tularemia patients. Culture of a draining primary lesion or an ulcer should be straightforward, provided effort is directed, using aspiration as necessary, at selectively obtaining deeper suppurative material. It is possible that excision of involved nodes may be associated with a constitutional reaction,[19] and exacerbation of systemic symptoms has been described as complicating incision of primary lesions as well.[2] An alternative method for culture of an infected bubo is to aspirate the node using a 20 gauge needle and a syringe containing 1 ml of sterile saline. Insertion of the needle into the bubo and aspiration until the fluid is blood-tinged (with or without injection of saline into the node) may produce satisfactory material.[10] In nonglandular disease, washings of the pharynx or stomach as well as sputum cultures have been used.[14] Blood cultures may also be indicated in seriously ill patients, although this procedure will go unrewarded in usual cases.[19]

The colonies resulting from our cases were initially small, resembling Haemophilus at 48 hours. They subsequently enlarged to become smooth white colonies (as opposed to the gray colonies usually described) of about 2 mm (Fig. 2), with the characteristic gram stain morphology.[19] The organisms can be identified with a specific agglutinin or with a fluorescent antibody technique. Our isolates were identified using the former technique and confirmed by the Centers for Disease Control in Atlanta.

5. Antibody titers have been the mainstay in the diagnosis of tularemia. It is likely that most laboratories rely on antibody titers for diagnostic purposes,[15] and the tests are well standardized and reliable at good reference laboratories.[10] A single titer of 1:40,[10, 19] 1:80,[4] and 1:160,[17] has been

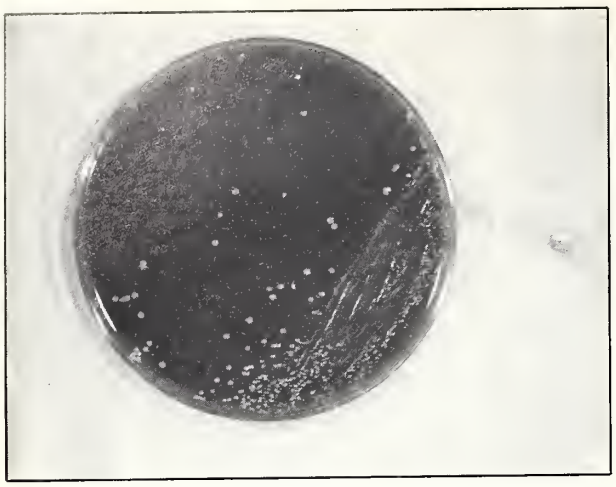

Figure 2
Enriched chocolate agar plate, showing 48 hour growth of Francisella tularensis.

described as being "diagnostic" in a compatible setting. Yet, as with other febrile agglutinin studies, a four-fold rise in titer is the hallmark of a diagnostic serologic test. This may not be accomplished until the convalescent phase, and acute serum should be saved to run in parallel with later convalescent studies. The height of the titer does not apparently correlate with the prognosis or symptomatology.[1] Two of the patients described here showed positive titers of 1:320 in the second week of illness which would be highly suggestive of the diagnosis, even as an isolated value. Serologic studies are not known to have been performed following positive culture and successful therapy in these patients or in Case 3.

A skin test is not widely available, but it has been reported to be positive in advance of a significant rise in antibody titer. It may be useful in identifying the diagnosis in as many as one-half of cases while still in the acute phase of the illness.[5]

Ordinary dye-stained preparations from smears of infected material are not likely to be diagnostically helpful. Fluorescent antibody techniques applied to such material or to tissue sections may be worthwhile,[19] but we have not used them in our laboratory.

SUMMARY

In conclusion, it is evident that physicians in this region may encounter cases of arthropod-borne ulceroglandular tularemia, and its symptom complex should be remembered. Cases of adenitis unresponsive to penicillin and without a positive culture for Streptococci or Staphylococci require further evaluation through culture or antibody studies, and treatment with streptomycin may be indicated. Laboratories may find that Francisella grows on present day enriched chocolate agar or other media; suitable precautions are in order when the organism is suspected or identified.

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About Our New President

HOWARD L. SAYLOR, JR., was born in Cogswell, North Dakota and moved to Huron, South Dakota shortly after World War I. He graduated from St. John's Military Academy, Delafield, Wisconsin. He then received a Bachelor of Science in Medicine degree from the University of South Dakota and his medical degree from Northwestern University, Chicago, Illinois. His internship, a rotating type, was completed at Northwestern Hospital, Minneapolis, Minnesota.

Dr. Saylor spent three years of active duty with the Army Medical Corp, serving in the European Theater of Operations. Following his discharge from the service, he went on to complete a surgical fellowship at the Mayo Foundation for Medical Education Research in Rochester, Minnesota in general surgery.

He has had an active practice in Huron since April 1, 1950. He is a member of the South Dakota State Medical Association, the American Medical Association, a diplomat of the American Board of Surgery and a Fellow of the American College of Surgeons.

He married Mary Ann Peterson of Huron and they have three children: Diane, a speech pathologist, married to Dr. Roger C. Wilson, oral surgeon, of Rapid City; Howard L. III has completed a surgical and vascular fellowship at the Mayo Clinic and is practicing in Minneapolis, Minnesota; and James C., a dentist, practicing in Watertown. This completes three generations of medical service, since Dr. Saylor's father was also an M.D.

South Dakota Society Of Pathologists

Officers for 1983-84

Jerry L. Simmons, M.D., President
John F. Barlow, M.D., Vice President
Beth L. Johnson, M.D., Secretary-Treasurer

President's Page

It is with a deep sense of humility and hope that I begin my year as your President for 1984. We are facing some rather trying times and certainly will be asked to sacrifice some of our rugged individualism in order to maintain, hopefully, high standards of delivery of medical service. It is only with the continued cooperation and support of our membership that we will be able to accomplish the direction of services that we feel is best for our patients and for medicine in general. During this time we need greater cooperation and united voice, both at the State and National level, than we have at anytime in the past. I am sure with the continuing dedication of our members of the Council and various commissions this can be accomplished. We do have a most capable staff in our office and I am sure that they will continue to help in all of the ways they have in the past.

I will endeavor to continue the high standards that have been set by my predecessors and hope that we can continue to provide good medical care, by the Doctors, for our patients.

Howard L. Saylor, Jr.

Howard L. Saylor, Jr., M.D., President
South Dakota State Medical Association

SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105

American Academy of Family Physicians

Report on Survey of 1983 Graduating Family Practice Residents

The total number of graduates surveyed was 2117. Of this number 1632 (77%) responded. Of these respondees, 1591 indicated the type of practice arrangement and 1363 specified the size of the community in which they plan to serve. A summary of the results as of July 1983 follows.

The data from 1977, 1978, and 1979 has been re-analyzed to conform with the 1980-1983 statistics. Caution must be exercised in comparing data previous to 1977 because of changes made to data analysis. However, the revised 1977-1979 data may be directly compared with the 1980-1983 data with confidence.

Practice Arrangements of 1983 Graduating Residents

Type of Practice Arrangements	Number of Reporting Grads	Percentage of Total Reporting Grads
Family Practice Group	336	21.1%
Multi-Specialty Group	154	9.7%
Two-Person Family Practice Group (Partnership)	245	15.4%
Solo	254	15.9%
Practice (arrangement not specified)	43	2.7%
Military	121	7.6%
Teaching	62	3.9%
USPHS	133	8.3%
Emergency Room	97	6.1%
Hospital Staff	25	1.6%
Research	4	.3%
Administrative	1	.1%
Further Training	30	1.9%
Fellowship	30	1.9%
None of the above	56	3.5%
	1,591	100.0%

Distribution of 1983 Graduating Residents by Community Size

Character and Population of Community	Number of Reporting Grads	Percentage of Total Reporting Grads	Cummulative Percentage of Total Reporting Grads
Rural area or town (less than 2500) not within 25 miles of large city	112	8.2%	8.2%
Rural area of town (less than 2500) within 25 miles of large city	26	1.9%	10.1%
Small town (2500-25,000) not within 25 miles of large city	342	25.1%	35.2%
Small town (2500-25,000) within 25 miles of large city	191	14.0%	49.2%
Small city (25,000-100,000)	246	18.1%	67.3%
Suburb of small metropolitan area	40	2.9%	70.2%
Small metropolitan area (100,000-500,000)	122	9.0%	79.2%
Suburb of large metropolitan area	146	10.7%	89.9%
Large metropolitan area (500,000 or more)	81	5.9%	95.8%
Inner city/low income area (500,000 or more)	57	4.2%	100.0%
	1,363	100.0%	

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This program has been reviewed and is acceptable for 16 prescribed hours by the American Academy of Family Physicians and 16 hours Category I AMA Physician Recognition Award.

WEDNESDAY, AUGUST 8, 1984

4:00 p.m. Board Meeting, SDAFP

THURSDAY, AUGUST 9, 1984

Lederle Day — Infectious Diseases

Lawrence W. Finney, M.D., Moderator

6:30-7:00 a.m. Registration
Complimentary Continental Breakfast
7:00-7:10 a.m. Welcome — L. W. Finney, M.D.
7:10-7:50 a.m. *Herpes*
James Blackman, M.D.
7:55-8:35 a.m. *Systemic Viral Infections*
Sheldon Landesman, M.D.
8:40-9:20 a.m. *Systemic Bacterial Infections*
Robert Marier, M.D.
9:25-9:45 a.m. Coffee, Conversation, Consultation
9:45-10:25 a.m. *Hepatitis B: Disease/Vaccine*
James Blackman, M.D.
10:30-11:10 a.m. *Pneumonia – 1984*
Sheldon Landesman, M.D.
11:15-11:55 a.m. *Antibiotics – 1984*
Robert Marier, M.D.
12:00-12:30 p.m. Panel with all Speakers
12:30-2:00 p.m. Luncheon for Registrants and Spouses, Hosted by Lederle Laboratories
Speaker: Mr. Jack Bowman, Vice President, American Cyanamid
2:00 p.m. Legislative Committee Meeting, SDAFP
Education Committee Meeting, SDAFP

AFTERNOON & EVENING FREE

FRIDAY, AUGUST 10, 1984

Surgery & Anesthesiology

Charles L. Swanson, M.D., Moderator

6:30-7:00 a.m. Registration
Complimentary Continental Breakfast
7:10-7:50 a.m. *Pre-operative Evaluation and Care*
Stuart Sybesma, M.D.
7:55-8:35 a.m. *Anesthetic Evaluation and Risks*
Donald Schellpfeffer, M.D.
8:40-9:20 a.m. *Post-operative Evaluation and Care*
Stuart Sybesma, M.D.
9:25-9:45 a.m. Coffee, Conversation, Consultation
9:45-10:25 a.m. *Regional Anesthesia Techniques*
Donald Schellpfeffer, M.D.
10:30-11:10 a.m. *Ileostomy/Colostomy Care*
Stuart Sybesma, M.D.
11:15-11:55 a.m. *Management of Intractable Pain*
Donald Schellpfeffer, M.D.
12:00-12:30 p.m. Panel with all Speakers
12:30-2:00 p.m. Hosted Buffet Luncheon for Registrants
SDAFP Annual Meeting

AFTERNOON & EVENING FREE

SATURDAY, AUGUST 11, 1984

Squibb Day: Cardiovascular Medicine & Nephrology

Michael Brown, M.D., Moderator

6:30-7:00 a.m. Registration
Complimentary Continental Breakfast
7:10-7:50 a.m. *Streptokinase*
C. Pat O'Brien, M.D.
7:55-8:35 a.m. *Congestive Heart Failure*
Phillip Hoffsten, M.D.
8:40-9:20 a.m. *Percutaneous Angioplasty*
C. Pat O'Brien, M.D.
9:25-9:45 a.m. Coffee, Conversation, Consultation
9:45-10:25 a.m. *Peripheral Vascular Disease*
Dean Borth, M.D.
10:30-11:10 a.m. *Drugs and the Kidney*
Phillip Hoffsten, M.D.
11:15-11:55 a.m. *Unstable Angina*
C. Pat O'Brien, M.D.
12:00-12:30 p.m. Panel with all Speakers

SEMINAR CLOSES

MAKE PLANS TO ATTEND NOW. WRITE: BLACK HILLS SUMMER SEMINAR
c/o South Dakota Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104

In this, the first of my "official" messages to the **South Dakota Journal of Medicine,** let me express my sincere appreciation to the South Dakota State Medical Association Auxiliary for the honor and privilege to serve as its President. A vote of thanks is extended to our past president, Marie Hovland (James), for her fine year of leadership and for her aid to me in preparing for the year ahead.

Let me take you back nearly two (2) years ago, some 2,000 miles away from home on vacation, when my current position became a reality. Our telephone rang and on the line was Ila Lushbough (Bruce), a member of the Nominating Committee. Well, after exchanging polite pleasantries, Ila asked me if I would consider the position of President-Elect to the South Dakota State Medical Association Auxiliary. I said: "WHAT?!! I had just "retired" from four years as Secretary to the South Dakota State Medical Association Auxiliary. I asked Ila for some time to think about the commitment and she said she'd hold! All I could say was "ILA!!" The question she asked was not complicated, yet it was a most vital one. After further conversation, I agreed, and at the same time couldn't help but wonder what I was getting myself into.

A lot of time has elapsed since that phone conversation. Contemplating my first trip to the AMA Auxiliary in June, I felt as though I was armed with a new passport and an international air ticket, and about to strike out for the far lands for the first time. Needless to say, it was one of life's great adventures, as was my attendance at the National Confluence in October.

Today's world places demands on people's time and talents as never before. We need to have great faith in our auxiliary — its purposes, goals, expectations. YESTERDAY: The AMA Auxiliary was formed to encourage fellowship among doctors' families. TODAY: It is a driving force for good health among the people in the United States. TOMORROW: It will be the strength to keep medical practice and health care at high levels. YESTERDAY, TODAY, TOMORROW . . . the AMA Auxiliary needs you and your active support to continue its work.

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Mary Wrage

Mary Wrage, President
South Dakota State Medical Association Auxiliary

South Dakota State Medical Association 1984 Annual Meeting Sponsor

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Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocuranine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

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J-4044 January 1984

Twenty-Four Year Old Caucasian Female With Large Pelvic Mass

Marva Souder, M.D.*
Samir Abu-Gazeleh, M.D.†
Richard A. Jaqua, M.D.‡
Discussers

John F. Barlow, M.D.§
Editor

Case #1006 416

This 24-year-old para 2012 with a last menstrual period 2 weeks previously was admitted to Sioux Valley Hospital because of an enlarged uterus.

For two months the patient had had epigastric discomfort without associated gastrointestinal or urinary tract symptoms. For approximately two months she had felt an enlarged lower abdominal mass. Ultrasonography had indicated a uterus of 16 weeks gestational size with multiple fibroids. Myomectomy was being considered because she wanted more children. The patient had had two term deliveries and one ectopic pregnancy but no other significant hospitalizations or illnesses. Menstrual periods occurred every 28 days with five days flow utilizing an average four pads per day. Periods had been slightly heavier recently with some cramping.

PHYSICAL EXAMINATION: Alert, oriented, temperature 37°C, pulse 80 per minute and regular, respirations 16 per minute and regular, blood pressure 110 systolic and 74 diastolic, height 5′4″, weight 51 kilograms. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The heart was not enlarged but had a grade I (six grades) systolic murmur heard best at the left sternal border with no radiation. Examination of the breasts was negative. There was a nodular, nontender mass arising from the pelvis and extending to the umbilicus (16-17 weeks pregnancy size), probably attached to the uterus. The external genitalia and vagina were negative. The cervix and adnexae were unremarkable. The uterus was anterior. Examination of the extremities including peripheral pulses and neurologic examination were within normal limits.

LABORATORY DATA: Urinalysis — yellow, clear, specific gravity 1.017, pH 7.0, negative for protein, glucose, bilirubin, ketones trace, hemoglobin 2+; sediment wbc/hpf rare, rbc/hpf 7-10. Hemoglobin 11.4 gm/dl, hematocrit 36 vol% with normal red cell indices, total leukocyte count 4,000/mm^3 (4.0 × 10^9/L) with a differential of 68% segmented neutrophils, 26% lymphocytes, 3% monocytes, 3% eosinophils. Serum sodium 137 meq/L, potassium 3.1 meq/L, chloride 103 meq/L, bicarbonate 30 mm/L; lactic dehydrogenase 600 μ/L (normal 0-270 μ/L), alkaline phosphatase, asparatate aminotransferase (AST, SGOT), total bilirubin, phosphorus, uric acid, creatinine within normal limits; total protein 4.8 gm/dl (normal 5.5-8.3 gm/dl), calcium 7.5 mg/dl (normal 8.4-10.7 mg/dl), glucose 148 mg/dl, blood urea nitrogen 4 mgs/dl, cholesterol 120 mg/dl. An operation was performed and a 1750 gram 18 × 18 × 15 cm right ovarian mass was found and reported on frozen section as a dysgerminoma. What is the operation of choice? What steps should be taken for further management of this patient?

DR. SOUDER: The final decision for the appropriate surgical treatment for each case of dysgerminoma can only be made intraoperatively. The possibility of a germ cell tumor should be considered preoperatively when a young female presents with a solid abdominal and pelvic mass, especially if it appears to be rapidly growing. Determinations of serum human chorionic gonadotropin (HCG) by beta subunit assay and alphafetoprotein (AFP) preoperatively, while not ruling out dysgerminoma, could implicate other germ cell tumors.

* Resident in Obstetrics and Gynecology, Sioux Valley Hospital, Affiliated Program, USD School of Medicine, Sioux Falls, SD.
† Specialist in Gynecologic Oncology, Sioux Valley Hospital. Associate Professor, Director of Gynecologic Oncology, Department of Obstetrics and Gynecology, USD School of Medicine, Sioux Falls, SD.
‡ Professor and Chairman, Department of Laboratory Medicine, USD School of Medicine. Pathologist, Sioux Valley Hospital and Laboratory of Clinical Medicine, Sioux Falls, SD.
§ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital. Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

Once the diagnosis of dysgerminoma is made, the surgical treatment may be conservative or radical. If there is any question of the accuracy of the diagnosis, the conservative approach is followed, i.e., unilateral salpingo-oophorectomy (USO).

The conservative approach is considered when the patient is less than thirty and has a desire for continued fertility. This represents a significant proportion of the patients since 80-90% of women with a dysgerminoma are younger than thirty, the median age being 19.5 years. Before deciding on conservative surgery, the stage of the tumor must be determined.

Only stage IAi tumors that are 10 cm or less in maximum diameter and not adherent to surrounding tissues at any point are appropriately treated conservatively. The criteria for stage IAi are that 1) the tumor is confined to one ovary, 2) the capsule is intact, 3) there is no ascites and 4) peritoneal washings are negative for malignant cells. (Peritoneal washings from the pelvis, lateral gutters and subdiaphragmatic area must be obtained before exploring the abdomen and removing the tumor.) Thorough exploration of the pelvis and abdomen is performed to rule out a more advanced stage. The diaphragm, liver, intestines, omentum and all peritoneal surfaces are explored for evidence of metastases which, if present, indicate stage III disease.

Bivalve or wedge biopsy of the opposite ovary is performed with frozen section to rule out bilateral involvement which is present in 5-10% of cases and places the tumor into stage Ib. In nulliparous patients the biopsy is also important in ruling out a streak gonad or gonadoblastoma of the opposite ovary. Gonadoblastomas often give rise to dysgerminomas or other germ cell tumors of the ovary. There is a small but definite incidence of dysgerminomas developing in persons with gonadal dysgenesis especially 46 X,Y testicular feminization, pseudohermaphrodites with XO and XO/XY mosaicism. If a streak gonad is present with or without a gonadoblastoma, it should be removed as there is a 10-15% incidence of gonadoblastoma or germ cell tumor developing in these dysgenetic gonads.

Bilateral selective pelvic and para-aortic lymphadenectomy as well as omental biopsies are done. The route of spread of dysgerminomas is primarily via the lymphatics. Spread also occurs to adjacent intraperitoneal structures. The presence of disease in these areas must be ruled out.

If, on histologic section, the tumor is anaplastic, conservative surgery is not recommended. Anaplastic dysgerminomas are described as having ≥ 3 mitoses/hpf, a small amount of stroma and slight lymphocytic infiltration. These tumors behave more aggressively.

When the conservative approach is not appropriate, radical surgery is done. This consists of a total abdominal hysterectomy and bilateral salpingo-oophorectomy with resection of all identifiable tumor that can be accomplished safely. In addition to the lymphadenectomies, an omentectomy rather than simple biopsies is done.

Postoperatively for the conservatively managed patient, if all surgical specimens other than the primary tumor are negative, no adjunctive therapy is necessary. If, however, dysgerminoma is found in any of the specimens or if malignant cells are found in the washings, additional therapy is indicated. Dysgerminoma is unique among germ cell tumors in being highly radiosensitive. For disease limited to the pelvis, external pelvic irradiation is indicated; for positive para-aortic lymph nodes, external pelvic, mediastinal and supraclavicular irradiation is indicated and for extranodal spread intraperitoneally, total abdominal external irradiation followed by a pelvic boost with shielding of the liver and kidneys after 1500 rads are the treatments of choice.

The need for adjunctive therapy may also become apparent after complete histologic evaluation of the tumor. Fifteen to twenty percent of dysgerminomas contain other germ cell tumor components. If other components are present, chemotherapy is indicated as the other germ cell elements are generally not radiosensitive. On the other hand, dysgerminoma does respond to chemotherapy, but the presence of other components worsens the prognosis. The overall five year survival with a pure dysgerminoma is about 80%. This decreases to about 31% with a mixed tumor. The corresponding five year survival with stage IA is 94% for pure dysgerminoma and 27% for dysgerminoma mixed with other germ cell elements. Positive preoperative AFP and HCG titers may point to a mixed germ tumor. Dysgerminoma may predominate in the tumor and a search for other components is mandatory by multiple histologic sections if the HCG or AFP levels are elevated. These levels may be followed to monitor the progress of therapy and point to recurrences or metastases. After 6-12 months of chemotherapy, the patient may be a candidate for a "second look" procedure to determine the need for continuing chemotherapy.

Survival in all stage I (confined to one or both ovaries) pure dysgerminomas is 90% at 5 years after surgery alone and 80% at 10 years. For stage IA (confined to one ovary) it is 95% and 92% at 5 and 10 years respectively, but drops to 50% for stage IIB (spread to other genital organs in the pelvis) at 5 years.

The recurrence rate overall for pure dysgerminoma is 25-35% — 17% for stage IA. Eighty percent of recurrences are within the first 2 years after initial

therapy with 75% in the first year. Because the majority of recurrences are within 2 years, the closest follow-up is in this period. Follow-up consists of a thorough physical examination with a good pelvic examination every 3 months for 24 months. The patient is then examined every 6 months for life.

If there are recurrences, the patient undergoes a metastatic work-up including possible surgery for confirmation and tumor resection as possible. If there is no history of radiation therapy, areas of recurrent disease are irradiated. If radiation therapy has been previously used or if the recurrent tumor has other germ cell components, chemotherapy is used. The survival for recurrent disease is 43.5% overall and 60% if the initial stage was IA. Recurrences have been found more than 10 years after initial treatment; thus, patients require lifetime follow-up. Also, it is recommended that conservatively treated patients have the other ovary, tube and uterus removed when they have completed their family.

Dr. Souder's Diagnosis
Dysgerminoma

*DR. K. G. PETERSON: I would like to show the sonographic study demonstrating a large solid, irregular inhomogeneous midline mass. There are scattered small cystic areas. The mass measures 14.5 cm in length and 16.0 cm in width. It is hard to determine the site of origin (Fig. 1).

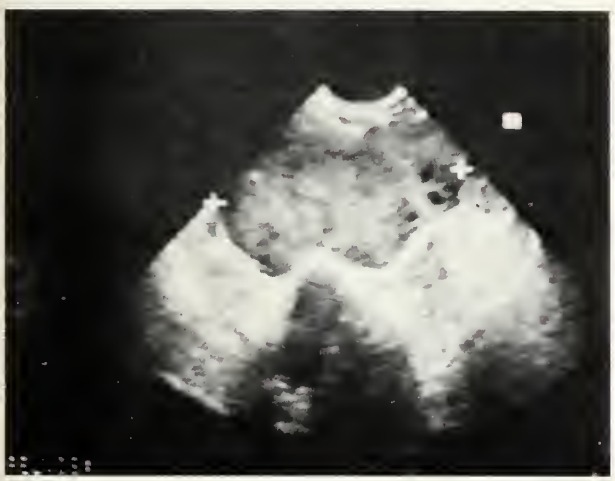

Figure 1
Sonogram with irregular mass in upper portion of picture showing dark cystic area (side with wide square and cross).

DR. SAMIR ABU-GHAZALEH: I would like to complement Dr. Souder on a nice discussion. I would like to point out that in a young woman such as this who has recently had a pregnancy and is found to have a solid ovarian mass, a fibroid (leiomyoma) is not the likely explanation. The patient would most likely have a solid ovarian tumor over 50% of which are malignant. 15% of these patients with malignant ovarian tumors will present with gastrointestinal symptoms. Another 15% have irregular menses but this can also be seen with fibroids. In my experience almost all young patients with malignant ovarian tumors have had some sort of gastrointestinal symptoms for some time prior to diagnosis.

Another possible clue to the diagnosis of malignancy in this case was the elevated lactic dehydrogenase (LD). Since one should have a high suspicion of malignancy in the ovary in this clinical situation, I believe tumor markers such as HCG and AFP should be performed preoperatively. Elevated levels of these would necessitate extensive sectioning of the tumor to rule out other germ cell elements.

Since patients with malignant ovarian tumors are often operated on by physicians other than gynecologic oncologists, my advice is that if there is no evidence of metastatic disease within the pelvis, simple removal of the tumor and waiting for the results of histologic examination of the ovarian lesion is the method of choice. This also applies if there is any hesitancy on the part of the pathologist in making a definitive diagnosis by frozen section. In this particular patient there were several findings which suggest a guarded prognosis. First of all, the tumor was large (over 10 cm. in diameter). Secondly, the tumor was adherent to the sacrum. After both frozen and permanent sections of these sacral adhesions, it was noted that there was no malignancy in the area of the adherence, but the tumor had simply grown too large and become stuck to the posterior wall as it rose out of the pelvis.

Staging of these lesions is very important. The diagnosis of stage IA cannot be made without: 1) a negative biopsy of the other ovary 2) extensive histologic examination of the omentum 3) histologic examinations of pelvic and paraaortic lymph nodes and 4) abdominal exploration. Only if all of these findings reveal no tumor, can the patient be classed as IA. I commend the pathologists in this case for suggesting deeper sections on the lymph nodes after initial examination. The deeper sections revealed metastatic carcinoma. I must also emphasize that the lymph node biopsy should include multiple lymph nodes. Examination of a single lymph node may easily miss tumor.

Since there is no large definitive series of dysgerminomas from even the major medical centers, the treatment of all patients with dysgerminomas must be individualized.

Since this patient had a pure dysgerminoma of the

* Pathologist, Sioux Valley Hospital and Laboratory of Clinical Medicine, Assistant Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

ovary, radiotherapy was the treatment of choice. Since the patient also had malignant cells in peritoneal washings, total abdominal radiation including the pelvis, paraaortic lymph nodes up to the diaphragm was indicated. We did not radiate the patients mediastinum or supraclavicular region. It is extremely controversial as to whether to do this but I feel with the advent of computer tomography (CT) we can now obtain serial studies with this excellent diagnostic instrument and treat the patient later should recurrence be noted. Certainly, if radiation had been performed above the diaphragm, encompassing the entire region of the axial skeleton, a substantial portion of the patient's bone marrow would have been included in the radiation portals. This might preclude further therapy if lung nodules should arise in the future. This is not to mention possible complications that might occur in the heart, mediastinum or lungs secondary to radiation.

It has been mentioned since recurrences are most likely to occur within the first year or two after initial treatment, close follow-up must be maintained. I see these patients every month for the first year and every two months during the second year. Chest x-ray and CT scans are done periodically for possible recurrence. These preferably should be done at the same institution so that proper comparisons can be made.

In patients who have had a positive cytology, but negative lymph nodes and omental biopsies in conjunction with a unilateral oophorectomy, triple chemotherapy has been used. Several patients so treated are living and well after such therapy. If the patient has retroperitoneal lymph nodes or has peritoneal metastases, they are classified as stage III. Since cancericidal doses of radiation are difficult to deliver to the upper abdomen especially if shielding of the liver or spleen is attempted, triple chemotherapy can be used. VAC (vinblastine, doxorubicin (adriamycin) and cyclophosphamide) or MAC (methotrexate, doxorubicin (adriamycin) and cyclophosphamide). Conservative therapy is not indicated in any patient beyond Stage IA. A total abdominal hysterectomy and bilateral salpingo-oophorectomy is indicated. Patients can live productive lives even though they cannot have children. Patients with stage III disease such as our patient today have at least a 75% five year survival. Since the patient had no gross tumor, I would think the survival should exceed that figure.

*DR. TOM GAECKLE: Were studies for AFP and HCG done in this case?

DR. SAMIR: Both levels were drawn after surgery and were negative.

DR. BARLOW: We received a 1750 gm mass measuring 18 × 18 × 15 cm. The surface was glistening tan and lobular with focal hemorrhagic areas and a thin fibrous capsule. Cut section was solid tan with focal cystic degeneration and hemorrhage (Fig. 2). Microscopic examination revealed a homogeneous cellular pattern composed of large nests of cells interspersed with a variable amount of hypocellular stroma with scattered lymphocytes. No granulomas were seen in the stroma. The tumor cells had round relatively uniform nuclei with prominent nucleoli and abundant clear cytoplasm containing glycogen demonstrated by special stains. There was no necrosis. In some areas mitoses were abundant (2-3 per high power field) but in other areas were sparse. The pattern is that of a classic germinoma often called dysgerminoma when it is found in the overy and seminoma when it is found in the testis (Fig. 3). Multiple sections were made to search for elements of choriocarcinoma, embryonal carcinoma, mature or immature teratoma or endodermal sinus tumor but none of these elements were found. No syncytial giant cells sometimes found in these lesions were seen.

DR. JAQUA: The electron microscopy on this case showed a typical germinoma. The cell cytoplasm was loaded with glycogen and there was a prominent irregular nucleoli in the nucleus commonly seen in this entity (Fig. 4).

Figure 2
Cross Section of tumor with lobulated appearance surrounded by thin fibrous capsule and area of hemorrhagic cystic degeneration.

* Specialist in Internal Medicine, Sioux Valley Hospital, Clinical Faculty, USD School of Medicine, Sioux Falls, SD.

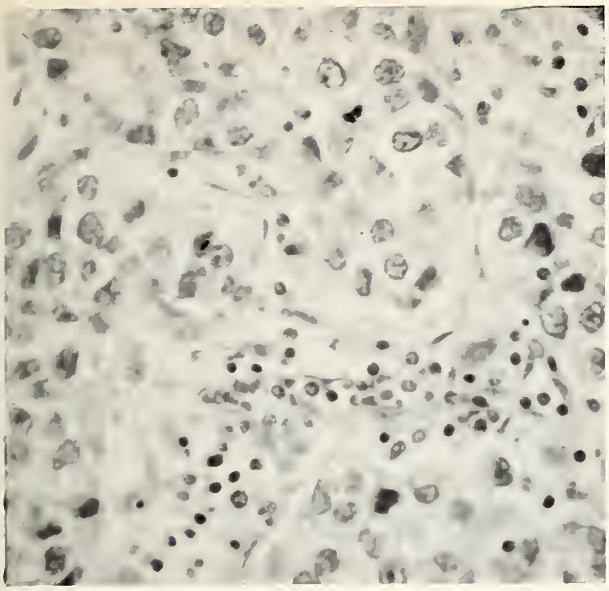

Figure 3
Nests of cells with uniform round nuclei and stroma with scattered lymphocytes. (H & E 400 ×)

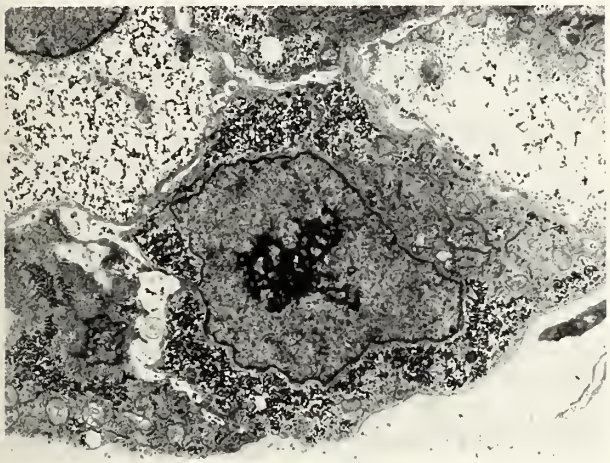

Figure 4
Typical cell of germinoma described in text as shown by electron microscopy.

DR. BARLOW: I should mention that a peritoneal washing was positive for malignant cells and a large paraaortic lymph node contained foci of metastatic germinoma. The omentum, other paraaortic lymph nodes, bilateral obturator, epigastric and common iliac lymph nodes were negative for tumor. The uterus, tubes and left ovary showed no remarkable findings.

FINAL ANATOMIC DIAGNOSIS
GERMINOMA ARISING FROM RIGHT OVARY

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Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Friday, April 13, 1984, in Sioux Falls, South Dakota. Following are items of business transacted at this meeting.

1. SEATED NEW COUNCILORS AND ALTERNATE COUNCILORS. New Councilors seated included C. D. Monson, M.D. from the Mitchell District, Edward James, M.D. from the Black Hills District and Jay Hubner, M.D. from the Yankton District. Alternate Councilors seated were Thomas Krafka, M.D. from the Black Hills District and John Sternquist, M.D. from the Yankton District.
2. 1984-85 BUDGET. The Council approved the budget as proposed. Two items were noted: 1) Provide an instate travel allowance for the president to reimburse him $.25 per mile not to exceed $2,000 per year. 2) Authorized the purchase of a new Association car for the Executive Secretary.
3. RECOMMENDATIONS TO THE GOVERNOR REGARDING MEDICAID RECIPIENTS AND HEALTH CARE. At the request of the Governor an Ad Hoc Committee of SDSMA physicians was appointed to study problems relating to providing health care to Medicaid recipients including cost and to submit recommendations regarding these problems. Eight recommendations were approved by the Council and submitted to the Governor for his consideration and any action he desires.
4. CODE FOR PHYSICIANS AND ATTORNEYS. The Council approved a Code for Physicians and Attorneys which was developed by the Medical-Legal Committee consisting of physician members of the SDSMA and members of the SD Bar Association. Pending approval from the Bar Association this will be sent to all SDSMA members.
5. PROFESSIONAL REVIEW ORGANIZATION CONTRACT FOR SOUTH DAKOTA. The Council approved the PRO proposal submitted by the South Dakota Foundation for Medical Care.
6. HONORARY LIFE MEMBERS. The Council elected the following to honorary life membership in the SDSMA: B. R. Skogmo, M.D., Mitchell; B. T. Otey, M.D., Flandreau; James Cline, M.D., North Carolina; Emil Hofer, M.D., Huron; and John Bailey, M.D., Rapid City.
7. SOUTH DAKOTA MEDICAL SCHOOL ENDOWMENT BOARD OF DIRECTORS. The Council reappointed Doctors Joseph Hamm, Warren Jones, G. E. Tracy, Bruce Lushbough, T. H. Sattler, Robert Giebink, and Bruce Allen to a one year term on the Board of Directors.
8. SODAPAC BOARD OF DIRECTORS. The Council appointed Michael Pekas, M.D., James Ryan, M.D., Mrs. Ellen Kunz, Duane Reaney, M.D. and Mrs. Kay Reaney to three year terms on the SoDaPAC Board of Directors.
9. AMA'S BENJAMIN RUSH AWARD. The Council voted to submit G. Robert Bartron, M.D. as a nominee for the Benjamin Rush Award presented annually by the American Medical Association.
10. ONE YEAR FREEZE ON PHYSICIANS' FEES. The Council voted to call for a one year freeze on physicians' fees in South Dakota. Notice of this was sent to all SDSMA members and to the news media.
11. MEDIA CODE OF COOPERATION. The Council approved a Media Code of Cooperation which was developed in conjunction with representatives of the Hospital and the State Medical Association. This will be made available to SDSMA members in the near future.

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Future Meetings

July

Advanced Trauma Life Support, Orr Major Hall, U. of Kans., Kansas City, KS, July 26-27. Fee: $350. 16 hrs. AMA Category I & 15 hrs. AAFP credits. Contact: Jody Scott, Dept. of Surg., U. of Kans. Med. Ctr., 39th & Rainbow, Kansas City, KS 66103. Phone: (913) 588-6124.

Pediatric Orthopaedic Surgery, Hyatt Regency, Minneapolis, MN, July 30-Aug. 1. Fee: $350. 18 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

August

Third Annual KU Summer Medical Symposium: Medicine for the Practicing Physician, The Broadmoor Hotel, Colorado Springs, CO, Aug. 2-5. Fee: $325. 12 hrs. AMA Category I & AAFP credits. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

Requested Topics for Clinicians, Marriott Hotel, New Orleans, LA, Aug. 3-5. Fee: $225. 11 hrs. AMA Category I credits. Contact: Dennis M. Oschipinti, M.D., 5640 Read Blvd., Ste. #400, New Orleans, LA 70127.

Pediatric Sports Medicine, Aspen, CO, Aug. 4-5. Contact: U. of Colo. School of Med., Off. of Postgrad. Med. Ed., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

27th Annual Pediatric Program, Aspen, CO, Aug. 6-9. Contact: U. of Colo. School of Med., Off. of Postgrad. Med. Ed., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

10th Annual Primary Care Orthopedics, Aspen, CO, Aug. 9-12. Contact: U. of Colo. School of Med., Off. of Postgrad. Med. Ed., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

Primary Care of the Back, Aspen, CO, Aug. 12-14. Contact: U. of Colo. School of Med., Off. of Postgrad. Med. Ed., 4200 E. 9th Ave., Box C265, Denver, CO 80262. Phone: (303) 394-5241.

Kidney Disease, Hypertension and Disorders of Fluids and Electrolytes, Aspen, CO, Aug. 14-18. Contact: U. of Colo. School of Med., Off. of Postgrad. Med. Ed., 4200 E. 9th Ave., Box C295, Denver, CO 80262. Phone: (303) 394-5241.

September

XV International Congress of the International Academy of Pathology, Fontainebleau Hilton, Miami Beach, FL, Sept. 3-7. Contact: Congress Secretariat, US-Canada Div. of Interna. Acad. of Path., 1003 Chafee Ave., Augusta, GA 30904. Phone: (404) 724-2973.

Advanced Trauma Life Support, Orr Major Hall, U. of Kans., Kansas City, KS, Sept. 6-7. Fee: $350. 16 hrs. AMA Category I & 15 hrs. AAFP credits. Contact: Jody Scott, Dept. of Surg., U. of Kans. Med. Ctr., 39th & Rainbow, Kansas City, KS 66103. Phone: (913) 588-6124.

Special Workshop on the Use of the CO_2 Laser in ENT Surgery, Sudler Hall, U. of Kans., Kansas City, KS, Sept. 6-7. Fee: $600. 16 hrs. AMA Category I & 15 hrs. AAFP credits. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

Conference on Homeostatic Functions in the Elderly, Sheraton Hotel, St. Louis, MO, Sept. 11-12. Contact: Conf. Sec., Geriatric Ctr., VA Med. Ctr., St. Louis, MO 63125. Phone: (314) 894-4659.

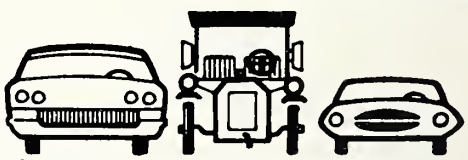
Leukemia Society Annual Symposium, Alameda Plaza Hotel, Kansas City, MO, Sept. 18. AMA & AAFP Category I credits available. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

American Society of Internal Medicine's Annual Meeting, Hilton Palacio Del Rio Hotel, San Antonio, TX, Sept. 20-23. Contact: Barbara Lauter, ASIM, 1101 Vermont Ave., NW, Ste. 500, Washington, DC 20005. Phone: (202) 289-1700.

Rehabilitation and Continuing Care in Cancer, Doubletree Hotel, Overland Park, KS, Sept. 27-28. AMA Category I & AAFP credits available. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

October

The Ninth Annual International Body Imaging Conference, Royal Lahaina Hotel, Maui, HI, Oct. 13-21. Fee: $425. 28 hrs. AMA Category I credits. Contact: Conf. Sec., Ninth Ann. Body Imaging Conf., Dept. of Rad., West Park Hosp., 22141 Roscoe Blvd., Canoga Park, CA 91304. Phone: (818) 340-0580, x280.



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Published Monthly by the S.D. State Medical Assn.

Volume XXXVII July 1984 Number 7

SOUTH DAKOTA JOURNAL OF Medicine

Clinicopathological Conference Eighteen Year Old Caucasian Female With Left Upper Quadrant Mass

Syndrome of Inappropriate Secretion of Anti-Diuretic Hormone and One Case Report

South Dakota's Family Practice Residency: A Ten Year Old Success Story

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References:
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BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dystunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

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SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 7 July 1984 (ISSN 0038-3317)

Official Publication of the
South Dakota State
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of The American Academy
of Family Physicians

the South Dakota Chapter
of the American College
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Eighteen Year Old Caucasian Female With Left Upper Quadrant Mass

Michael E. Hogue, M.D.*
Donald B. Graham, M.D.†
Richard A. Jaqua, M.D.‡
Discussers

John F. Barlow, M.D.§
Editor

CASE #1 019 184 (83-6273)

This 18 year old, caucasian, unmarried female was admitted to Sioux Valley Hospital because of a left upper quadrant mass.

Eighteen months prior to admission the patient consulted a physician in Miller, South Dakota because of a fever to 38.5°C and sore throat. Her physician noticed she had enlarged tonsils and mild pharyngitis. She was treated with penicillin and her fever decreased but she had intermittent fever without chills or localizing signs except for mild intermittent left sided nondescript upper abdominal pain approximately every other month. Investigation of her urinary tract for suspected infection revealed a negative intravenous pyelogram (IVP) and urine culture. Several different antibiotics did not abolish the febrile pattern. Two months prior to admission on a routine physical examination a left upper quadrant mass thought to be due to enlargement of the spleen was noted.

PHYSICAL EXAMINATION: Alert, cooperative, young woman, pulse 64/minute and regular; temperature 37°C, respirations 16/minute and regular; blood pressure 100 systolic and 62 diastolic, weight 63 kg. Examination of the head and neck was unremarkable. The chest was clear to auscultation and percussion. The heart was not enlarged and had a regular rhythm with no abnormal murmurs or extra sounds. The breasts were negative. There was no significant axillary or cervical lymphadenopathy. On abdominal examination there was minimal point tenderness in the left upper quadrant. There was a left upper quadrant mass which came downward on deep inspiration and was felt to be the spleen. Pelvic examination was unremarkable and there were no adnexal masses. Neurologic examination and examination of the extremities revealed no abnormalities.

LABORATORY DATA: Urinalysis, yellow, clear, specific gravity 1.008, pH 5.5, negative for protein, glucose, ketones, bilirubin and hemoglobin; sediment — negative. Admission hemoglobin 13.5 gm/dl, hematocrit 40 vol/dl with normal red cell indices. Total white count 6.4 $\times$ 10^9/L with a differential of 35% segmented neutrophils, 57% normal lymphocytes, 6% monocytes, 1% eosinophils and 1% basophils. A platelet count was 191 $\times$ 10^9/L. A reticulocyte count was 1.2% and erythrocyte sedimentation rate was 1 mm per hour. A fluorescent antinuclear antibody test (FANA) was negative; sodium 139 meq/L, potassium 3.8 meq/L, chloride 109 meq/L, CO2 content 24 mm/L; lactic dehydrogenase (LD), alkaline phosphatase, aspartate aminotransaminase (AST), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. A protein electrophoresis was within normal limits. A T4 (thyroxine) and thyrotrophin (TSH) were within normal limits. An amylase was 104 μ/l (normal 0-74 μ/l). A gamma glutamyl transpeptidase (GGPT) was within normal limits. A monotest was negative. An antistreptolysin (ASO) was 170 Todd units (normal 0-150 Todd units). Titers for brucella and tularemia revealed no titer. IgM and IgG titers for Toxoplasma gondii, Ebstein Barr virus and Cytomegalovirus revealed no significant titer to Toxoplasma and evidence of remote infection to Epstein Barr virus and Cytomegalovirus. Urine and blood cultures for bacteria were negative. A chest film was normal. A liver and spleen scan showed no diagnostic abnormality. A sonogram revealed a 10 cm solid mass lying in the left upper quadrant which was separate from the spleen and possibly an accessory spleen. An abdominal computer tomogram (CT) revealed the left upper quadrant mass that was separate from the spleen and could be retroperitoneal or intraabdominal. Titers for Histoplasma capsulatum, Blastomyces

* Resident in Family and Community Medicine, Sioux Falls, Affiliated Program with School of Medicine, University of South Dakota, Sioux Falls, SD.
† General Surgeon, Sioux Valley Hospital, Clinical faculty, School of Medicine, University of South Dakota, Sioux Falls, SD.
‡ Professor and Chairman, Dept. of Laboratory Medicine, School of Medicine, University of South Dakota, Pathologist, Sioux Valley Hospital and Laboratory of Clinical Medicine, Sioux Falls, SD.
§ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital: Professor of Pathology, School of Medicine, University of South Dakota, Sioux Falls, SD.

dermatitidis, Coccidioides immitis, Aspergillus, and Candida were negative. An operation was performed.

DR. HOGUE: This is a very difficult case, but I will try to use it as an example of a clinical approach to abdominal masses. In spite of a variety of new imaging techniques, the history and physical examination are very important. One must elicit the type and location of the pain and its relationship to food. A history of anorexia, weight loss, change in bowel habits or stool habits would be quite important. A history of trauma might suggest a pancreatic pseudocyst. A history of previous surgery could certainly suggest bowel stricture or intestinal obstruction from adhesions. Knowledge of the previous pathology would certainly be extremely important. On physical examination, the location of the mass and any associated organomegaly would be important. The presence of guarding, tenderness or abdominal rigidity is paramount in clinical evaluation as well as the presence or absence of bowel sounds. Laboratory tests, which might be helpful, include examination of the stool for gross or occult blood, a hemogram to indicate whether iron deficiency anemia (blood loss) was present and a white count since an elevation with a shift to the left might suggest infection such as an abscess. A urinalysis looking for the presence of red cells might indicate renal pathology. One should certainly obtain blood urea nitrogen, creatinine and electrolyte determination to evaluate renal disease. Liver function tests would be indicated. In this particular case, serum and two hour urinary amylase levels would be important to evaluate possible pancreatitis or pancreatic neoplasm. An amylase-creatinine clearance ratio might be helpful as renal tubular absorption of amylase during pancreatitis has been said to be decreased. An alpha fetoprotein would be elevated in liver disease such as an hepatic carcinoma. A carcinoembryonic antigen might be elevated in metastatic carcinoma to the liver. One could obtain a test for heterophil antibodies (monospot) to suggest the presence of infectious mononucleosis as the intra-abdominal mass was suspected to be an enlarged spleen. However, I would not expect the spleen to have been large for such a long time in infectious mononucleosis. This test was negative in this case.

There are a wide variety of new imaging studies which could be useful in evaluating any abdominal mass. These include flat plate of the abdomen, barium enema studies, intravenous pyelogram, ultrasound, computer tomogram, radionuclide liver and spleen scan as well as endoscopy-endoscopic retrograde cholangiopancreatography (ERCP). In this case, the ultrasound and computer scan were perfectly adequate to evaluate the lesion.

There is a large variety of neoplasms which this mass could represent. I believe a neoplasm of the esophagus would be quite unusual in this case. There are a variety of benign and malignant neoplasms of the stomach, but a carcinoma or lymphoma could produce this abnormality. The same might be said about similar neoplasms of the small bowel or a carcinoid of the small bowel, but the lack of gastrointestinal symptoms would rule most of these out. There is, also, no evidence of carcinoid syndrome. Neoplasms of the large bowel would be unlikely in this situation without any indications of bleeding or obstruction. A neoplasm of the mesentery, peritoneum or diaphragm such as a mesothelioma could produce this abnormality, but these are rare. A peritoneal mesothelioma is usually associated with asbestosis. Large tumors from one of the above structures including a leiomyoma or leiomyosarcoma are possible. A hemangiopericytoma is another possibility. The radiologist felt he could separate this lesion from the kidney but could not completely exclude a renal neoplasm such as a hypernephroma or Wilm's tumor. A renal cyst would be unlikely with this appearance on ultrasound and computer tomography. A neoplasm of the adrenal gland such as a large adenoma or carcinoma is a possibility. We have no evidence of steroid excretion. Ninety per cent of adrenal carcinomas do produce abnormal steroids and these have a tendency to invade the renal vein. Adenomas are rarely over 5 cm in diameter. We have no evidence of a pheochromocytoma. Neuroblastoma would be extremely rare in this age group.

The most likely location of this mass is the pancreas. Ectopic pancreas is usually only found at autopsy and measures 3-4 cm in maximum diameter. Occasional congenital retention cysts of the pancreas occur or a pseudocyst which follows pancreatitis or trauma may occur. Multilocular cystadenoma or cystadenocarcinomas can range from 5-15 cm and are certainly a possibility in this case. The malignant lesions can be locally invasive. However, only one-third of these neoplasms are in patients less than 50 years old although females do predominate in a 9 to 1 ratio. Pain is an infrequent symptom.

An adenocarcinoma of the pancreas would be very unusual in this age group and is more commonly seen in males. It is more common in smokers and two-thirds of the lesions occur in the head as opposed to the body and tail. The patients also usually have abdominal pain, anorexia and weight loss.

I suppose we cannot completely exclude an hepatic lesion in the left lobe of the liver. Congenital cysts of the liver and bile duct adenomas are possible. There is a condition called a mesenchymal hemartoma which can produce a mass which occurs in patients under 20 years old. Cavernous heman-

giomas of the liver are common tumors but they are usually less than 3 cm in diameter. Liver cell adenomas have been associated with the use of oral contraceptive agents in women 20-40 years of age but we have no evidence of this predisposing factor in this case. Hepatocellular carcinomas of the liver usually occur secondary to cirrhosis for which we have no evidence.

Since we have ruled out splenomegaly, I will not go into that differential diagnosis. I suppose that a neural soft tissue neoplasm such as neurofibroma, ganglioneuroma or other soft tissue neoplasm is possible in this case.

Dr. Hogue's Diagnosis
Pancreatic Neoplasm of Unknown Type

DR. GRAHAM: At operation there was a 10 cm mass arising from the distal body of the pancreas. The remainder of the pancreas, liver, gallbladder, spleen, large and small bowel, uterus, tubes, ovaries and kidneys were normal. A distal one-half pancreatectomy and splenectomy and catheter jejunostomy was performed. A frozen section diagnosis was that the lesion was an epithelial tumor, not a lymphoma but possibly malignant and possibly a pancreaticoblastoma.

DR. BARLOW: The mass in the body of the pancreas was 11.0 × 10.0 × 5.0 cm well circumscribed but with no true capsule. Cut surface was tan-yellow with focal areas of punctate red to blue discoloration with an eccentrically located 3.5 × 1.5 cystic space (Fig. 1).

The tumor had two major low power histologic patterns. One was solid sheets of cells interspersed by multiple thin-walled vessels. The second was a papillary architecture. The nuclei were uniform, round to oval with only a rare mitosis and inconspicuous nucleoli (Fig. 2). The cytoplasm was sparse and cell borders poorly defined. There were also microcystic areas containing PAS positive material and areas of fibrosis or myxoid change in the stroma giving the tumor the appearance of a mixed tumor of the salivary gland.

DR. JAQUA: Because of the solid endocrine-like appearance of this tumor and our initial uncertainty about the appropriate classification of this tumor, ultrastructural studies were performed. We were particularly interested in detection of any differentiation toward exocrine pancreas in which one would expect to see zymogen granules in the cell cytoplasm or endocrine differentiation in which we would expect to see neuroendocrine-like granules similar to those seen in the pancreatic islets. The samples for electron microscopy were from areas showing significant myxoid degeneration of the stroma. The cells were definitely epithelioid in nature with well developed basal lamina, polarized cytoplasmic architecture and moderately well developed Golgi apparatus at the apical pole of the nucleus. Only rare lumen-like areas were identified, some intracytoplasmic, with only poorly developed microvilli. Junctional complexes were rather poorly developed, and the cytoplasm was free of secretory granules of

Figure 1
Large mass with eccentric area of cystic degeneration. This is located in body of pancreas. Spleen can be seen toward bottom of picture.

Figure 2
Low power of tumor showing papillary and solid areas as well as hyalin stroma.

TABLE I
SUMMARY OF CASES OF SOLID AND PAPILLARY TUMORS OF PANCREAS

SOURCE	SEX	AGE	RACE	LOCATION	SURGICAL PROCEDURE	FOLLOW-UP
1. AFIP	F	20	unknown	tail	distal pancreatectomy	21 years, no recurrence
2. AFIP	F	24	black	tail	distal pancreatectomy	15 years, no recurrence
3. AFIP	M	2	unknown	head and body	Whipple	operative death, hemorrhage
4. Hammoundi et al	F	12	black	head	Whipple	16 months, no recurrence
5. Boor and Swanson	F	19	black	tail	distal pancreatectomy	1 year, no recurrence
6. Schlosnagle & Campbell	F	25	black	tail	distal pancreatectomy	
7. Schlosnagle & Campbell	F	21	white	head	head of pancreas pancreaticoduodenectomy	
8. Cubilla & Fitzgerald	F	12	unknown	body and tail	partial pancreatectomy	recurrence at 7 years, alive 9 years after first operation
9. This case	F	18	white	tail	distal pancreatectomy	

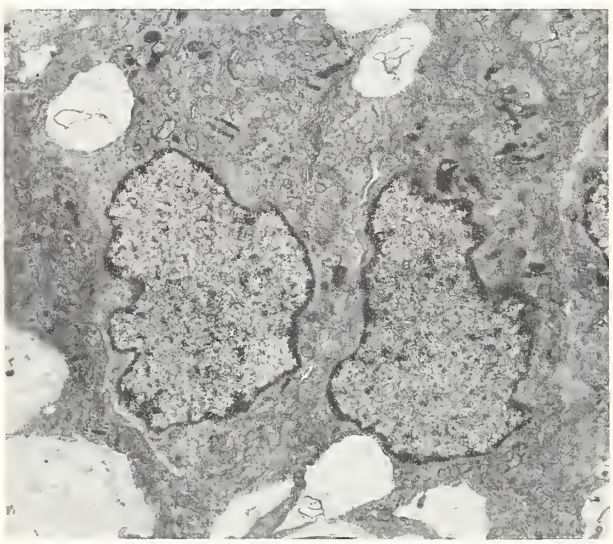

Figure 3
Ultrastructure of two epitheloid cells from tumor devoid of zymogen or endocrine granules and suggesting ductal origin.

endocrine, exocrine or mucin type. Many of the cells contained rather abundant mitochondria and scattered secondary lysosomes were present. The overall morphology was that of pancreatic duct origin. This agrees with the cases of papillary and solid neoplasms of the pancreas in childhood and young adults on which ultrastructure has been performed. One case in the literature[2] showed occasional neurosecretory granules which was interpreted as early differentiation toward pancreatic islets. The pancreatic ducts are felt to give rise to the islets in embryogenesis. (Fig. 3)

FINAL ANATOMIC DIAGNOSIS
SOLID AND PAPILLARY CYSTIC TUMOR OF PANCREAS

DR. BARLOW: Neoplasms of the pancreas in children are extremely rare. The most common lesions are functional and nonfunctional islet cell carcinomas. Ductal adenocarcinomas number only 22 in the world literature and are usually rapidly fatal.

The present tumor probably fits best into a rare group of tumors with solid, papillary and cystic areas. They present as abdominal masses with or without pain and are usually well circumscribed and fairly large. Although there are too few cases to be certain, prognosis may be good but metastases do not occur. The following table shows cases culled from the literature (Table I). According to Cubilla and Fitzgerald,[4] Oertel has collected 52 cases at the Armed Forces Institute of Pathology (AFIP) mostly in young females with metastases in only 2 cases.

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President's Page

Election year 1984 — of all the times in recent history this year can be pivotal to the future of medicine and delivery of health care in the United States. We now have the highest level of health care in the world, and we must do all we can to maintain this.

This Spring I had the privilege of attending the AMA Leadership Conference, and recently the House of Delegates of our sister state Minnesota. The problems are the same basically, and we will only continue our providing of health care with cooperation from the "grass roots" up. We must become politically involved at all levels, now and forever. We must maintain our high quality care; make available proper care for the elderly and medically indigent; assume leadership in "public education" of the how, why, and what in health care; and help educate the business community along the same lines.

In order to accomplish these goals, we must institute and continue a "one on one" dialogue with our municipal, county, state, and federal elected officials by direct contact, phone, and correspondence. We also have a built-in method of attention-getting through our SoDaPAC and AMPAC programs. All of these programs will only be as strong as the individual makes them. It may be a very trite saying "united we stand" but never more true than it is today.

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Howard L. Saylor, Jr., M.D., President
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Syndrome of Inappropriate Secretion of Anti-Diuretic Hormone and One Case Report

Neyton Baltodano, M.D.*

ABSTRACT

Hyponatremia and inappropriate secretion of sodium in the urine is one of the most common clinical findings in the chronically ill elderly patient. This is a review of the syndrome and the most common causes, as well as a report of a case related to the intake of one of the most common anti-depressants, Tofranil® (imipramine). The study was followed for 7 days after the syndrome was established with the control of sodium in the blood and urine and the correlation with the clinical picture. Patient responded well to the water deprivation and to the stop of the medication. The case suggested that some medications in the elderly patient, even if he is not seriously ill, could lead to SIADH. It is also important to note that sometimes not well worked up elderly patients may get the wrong diagnosis when he changes the mental status.

SIADH

The syndrome of inappropriate secretion of anti-diuretic hormone has been known for many years, since 1967, when Bartter describes it as a syndrome with low sodium in the blood and a high concentration in the blood stream of the anti-diuretic peptides, with all the neurological manifestation due to brain edema. It is known too, that renal excretion of the water is impaired because the patient continues to drink the accustomed amount of water producing dilution and expansion of the body fluid. The most common causes of this syndrome are known to be related to malignant tumors, one of them the oat cell carcinoma of the lung; also infections which produce a stress the patient reacts to, producing this high amount of anti-diuretic hormones. Other causes include brain tumors and traumas, myxedema and drugs including sulfonylureas, thiazides, anti-depressants and anti-tumor agents. (Table I).

TABLE I
CAUSES

- I. Ectopic
  - a. CA of Lung
  - b. CA of GI Tract
  - c. Pancreas CA
  - d. TB
  - e. Lymphoma
- II. Supra Optic Hypophysis Secretion
  - a. Trauma of Head
  - b. Pneumonia
  - c. Hemorrhage
  - d. CNS disease
  - e. Addison's Disease
  - f. Idiopathic
- III. Drugs
  - a. Chlorpropamide
  - b. Carbamazepine
  - c. Amitriptyline
  - d. Vincristine

In order to make the diagnosis of SIADH the criteria should be: 1) that extracellular fluid osmolality in sodium concentration must be decreased, 2) the urine must be hypertonic to serum, 3) sodium excretion in the urine continues to exceed 10 meq per liter despite hyponatremia, 4) adrenal, renal and

* Internal Medicine, Kingsbury County Hospital, Lake Preston, SD.

cardiac function must be normal, 5) the hyponatremia must be corrected by water restriction.

Studies by Deucth et al 1966 show that in elderly patients normally evaluated previously to surgery the syndrome was not related to any drugs they used during the surgery. Recent reports by Elderman show that there must be an increased susceptibility of the older patient to develop hyponatremia, despite the health condition of them.

In 1979, Klienfield et al showed that from 160 chronically ill patients, 36 of them developed this syndrome. It has been known for many years that the elderly patient may have a volume baroceptor receptiveness which decreases compared with a young subject.

The relation between anti-depressants and SIADH has not been extensively studied in psychiatric patients taking different medications (Baltodano et al 1980). It is interesting that there have been only two cases reported in 1974 by Luzecky, M., of patients taking Amitriptyline who developed this syndrome.

The physiopathology of this medication causing SIADH is not well known at the moment and there has not been a case of imipramine reported.

CASE REPORT

This is an 83 year old white male admitted to the hospital with chief complaints of dizziness and mild hypertension. In the history too, it is known he has been depressed and crying for the last three months. The previous physician did not give him any antidepressant medication. While he was admitted he had a complete work up for his brain, ENT, electrolytes, CBC, lumbar puncture and cardiac monitor. All of them were found to be normal except for the scan of the brain which showed mild brain atrophy. At admission, his electrolytes were: sodium 138, potassium 4.2, chloride 98, CBC was normal with an hemoglobin of 14.5, white blood count of 7000. EKG shows a sinus rhythm with a normal axis. Glucose was normal. Because of the depression, the patient was started with Tofranil® 25 mg three times a day; and 48 hrs after the patient acted disoriented and very confused. The neurological examination and the glucose were normal, but surprisingly, the sodium went down to 104. There were no signs of heart failure or of renal dysfunction. His creatinine was 1 mg, the BUN was 16, the urine was collected for 24 hrs to check the concentration of sodium and he was found to have more than 90 meq per liter. His total output was over 1000 ml in 24 hrs. The medication was stopped and the levels of Tofranil® were drawn and found to be normal. The water deprivation test treatment was started and after 7 days his electrolytes slowly increased by 3 to 5 meq per day and then normal levels of 138 meq per day after the eighth day of treatment. Patient recovered his mental status and the examination reported orientation × 3. Patient was discharged with the only restriction of fluids 500 cc per day for the next 7 days and monitoring of electrolytes. There was no need to give any other medication due to the good response to the water deprivation.

CONCLUSION

This is a case report of syndrome of inappropriate secretion of anti-diuretic hormone due to the use of tricyclic anti-depressant. The case was reported based on the criteria of the inappropriate secretion of antidiuretic hormone from what I mentioned before. It would be interesting if these cases were more worked up by the treating physicians in the nursing homes, the place where we usually see them.

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Distribution of USDSM Graduates in First Year Residencies by Type of Program
1977-84
An Eight Year Summary

Type of Program	U.S. Graduates (NRMP 8 yr avg) 1976-84* Percent	USDSM Graduates (8 yr summary) 1977-84 Number	Percent
Family Practice	13.0	79	22.0
Internal Medicine	35.5	97	27.0
General Surgery	13.0	31	9.0
Pediatrics	10.0	25	7.0
Obstetrics/Gynecology	6.0	21	6.0
Psychiatry	4.0	6	2.0
Medical Specialties	1.0	2	1.0
Dermatology		(1)	
Neurology		(0)	
Ophthalmology		(1)	
Surgical Specialties	3.0	10	3.0
Neurosurgery		(0)	
Orthopedics		(8)	
Otolaryngology		(0)	
Urology		(2)	
Plastic Surgery		(0)	
Support Specialties	7.5	19	5.0
Anesthesiology		(6)	
Emergency Medicine		(0)	
Pathology		(7)	
Physical Medicine		(6)	
Radiology		(6)	
Transitional	7.0	63	18.0
	100%	353†	100%

* Source: Journal of Medical Education, 53(6):500-502, 1978; 54(4):347-349, 1979; 55(4):382-384, 1980; 56(4):372-374, 1981; 57(5):420-422, 1982; 58(5):438-440, 1983.
† 94 of 353 (28%) entered USDSM affiliated residencies.

USDSM M.D. Graduates Entering Family Practice Residencies
An Eight Year Summary

Academic Year	Number Of Graduates	Number Entering FP Residencies	Number Entering SD FPR Program
1976-77	39	9(23%)	4/9
1977-78	36	4(11.1%)	4/4
1978-79	39	11(28.2%)	7/11
1979-80	47	14(30%)	2/14
1980-81	49	20(41%)	8/20
1981-82	44	6(13.6%)	1/6
1982-83	49	8(16.3%)	2/8
1983-84	50	7(14%)	4/7
	353	79(22%)	32*/79(40%)

* Six Residents left the program at the end of one year: 1 to enter another FP Residency, 4 to change discipline, 1 to enter practice.

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South Dakota's Family Practice Residency: A Ten Year Old Success Story

Earl D. Kemp, M.D.*

ABSTRACT

South Dakota's Family Practice Residency has recently celebrated its 10th anniversary. The history and accomplishments of the program are reviewed, and a brief look forward is described.

July of 1983 marked the tenth anniversary of the establishment of the Family Practice Center, Inc., South Dakota's only residency program in Family Practice. This occasion, which was celebrated in Rapid City by a reunion honoring Lloyd Sweeney, M.D., the Program's founding director, seems also to be an appropriate one for reviewing the residency's progress.

BACKGROUND

At a time when the specialty of Family Medicine was very new, and a move to eliminate the free standing rotating internship was gaining momentum, it also was shown that the need for family practitioners in the state of South Dakota was critical and increasing.[1] Both McKennan and Sioux Valley hospitals in Sioux Falls already had established internship programs and a continuing interest in training young physicians as well as other medical personnel. With the combination of the state's need for more primary care physicians, the hospitals' desire to continue to train physicians, and the need of the specialty of Family Medicine for more training sites, the decision was made by the boards of McKennan and Sioux Valley Hospitals to establish a non-profit corporation whose purpose was to train Family Practice residents.

Since the establishment in 1973 of the Family Practice Center, Inc. (FPC), under the directorship of Dr. Sweeney, and a charter graduating class of two residents, there has been continuing growth until July of 1980 when the program reached its present size of 12 resident positions in each of its three years. Sixty-six physicians have graduated from the program thus far. Under the direction of Robert Cloar, M.D., a more adequate level of faculty was recruited, and the sponsoring hospitals provided funds for the construction of the present model clinic which was occupied in the fall of 1981.

QUALITY

Throughout the program's existence, one of the prime concerns of the faculty has been the quality of teaching in the residency. Through continuing attention to the educational needs of residents, as well as to the guidelines of the several organizations whose requirements must be met, full accreditation has been maintained from the outset. On the basis of the most recent survey in April of 1983, the maximum possible 5 years of continued accreditation was awarded.

There are two methods presently available for comparison of the knowledge of South Dakota's residents to that of residents from other programs. South Dakota's trainees and residency graduates have demonstrated the quality of their education through their performance on both the In-Training Examination and on the Certification Examination of the American Board of Family Practice (ABFP). The In-Training Examination is given to virtually all Family Medicine residents across the country in each of the three years of training. The resulting data

* Director, Family Practice Center, Inc., Associate Professor and Vice Chairman, Department of Family Medicine, University of South Dakota School of Medicine.

permits comparison of both individuals and resident classes to other residents at similar levels of training. Residents in our program have consistently achieved higher average scores than the national norms in the various areas tested.[2] The ABFP also provides data to residencies on the performance of their graduates on the specialty certification examination. Of the last 24 FPC examinees, 46% scored in the top ⅓ nationally and 79% scored in the top ½ nationally, while even the lowest score achieved was a 45th percentile rank.[3, 4] Furthermore, since 1976 (the earliest year for which ABFP scores were released to our program) there has never been a year when the average certification scores of our graduates has not been higher than the national average.[4]

LOCATION OF GRADUATES

The charge of the Family Practice Residency is to adequately prepare young physicians to provide medical care to the many underserved areas of rural South Dakota, as well as other communities of the Upper Midwest, and thereby help relieve the health provider manpower shortage and maldistribution which has been recognized in this region of the country.

South Dakota communities which already are served by graduates of the Family Practice Residency are Brookings, Chamberlain, Custer, Deadwood, Dell Rapids, Ellsworth, Gettysburg, Gregory, Madison, Pierre, Rapid City, Sioux Falls, Spearfish, Wagner and Watertown. In addition, members of the 1984 graduating class will be locating in Canton, Hot Springs, and Milbank.

Presently more than half of FPC graduates practice within the state of South Dakota, and ¾ are in S.D. or the immediately contiguous states. (Table I)

Of those residency graduates located elsewhere, many are fulfilling obligations in the Indian Health Service, serving as medical missionaries etc.

COST

In addition to providing high quality training to our residents, South Dakota's Family Practice Program has accomplished its mission in a cost effective manner. While it is a fact that residency training is expensive, approximating an average cost of $42,000 per resident per year during the first 10 years of our program's existence, a 1982 University of Minnesota study revealed that the average cost of training Family Practice residents during that year among 25 midwestern programs was $56,739 per residency position, compared to $52,934 in South Dakota.[5] Furthermore, this cost in our program has increased an average of less than 5.7% in each of the past two years.

THE FUTURE

As is ordinarily the case, graduates of South Dakota's Family Practice Residency have initially been more likely to settle in and near the base community. However, again as expected, as the need in the Sioux Falls area is met, an increasingly higher percentage of graduates will locate in the other medically needy areas of the state and region. This trend is exemplified by the commitment of the present third year class, of whom 30% plan to locate in Sioux Falls, 50% in other South Dakota communities, and 20% in contiguous states.

A continued demand for at least the present number of graduates in Family Medicine will continue through the foreseeable future in South Dakota.[1] Although there has been some interest in developing either independent or related residencies in other communities, there are presently neither plans nor facilities for further expanding the present program in Sioux Falls. Nevertheless, because of the increasing financial squeeze on the hospitals, and with the waning of federal support for medical training programs, a need for modification in the financial sup-

TABLE I
DISTRIBUTION OF RESIDENCY GRADUATES

Year	S.D. — Not Sioux Falls	S.D. — In Sioux Falls	Contiguous States	Elsewhere	Total
1975	1	0	0	1	2
1976	0	0	3	0	3
1977	0	1	1	2	4
1978	4	1	3	0	8
1979	0	4	1	3	8
1980	3	2	2	1	8
1981	5	2	2	3	12
1982	8	1	0	3	12
1983	0	2	2	5	9
	21 (32%)	13 (20%)	14 (21%)	18 (26%)	66
1984 (to date)	5	3	2	0	10
	26 (34%)	16 (21%)	16 (21%)	18 (24%)	76

port base for the program is clearly emerging. New sources of funding will need to be actively cultivated, and some shift from federal to state support must also occur if the program is to continue at its present size.

CONCLUSION

The past 10 years have demonstrated the success of South Dakota's Family Practice Residency in fulfilling its mission in a quality, cost effective manner. The need throughout the state for appropriately trained, accessible physicians is being met, though there is still a long way to go to fully satisfy this need.

These accomplishments have been achieved with a great deal of support from the medical community throughout our state. With continuation of that support, the challenge will continue to be met. This will help assure that South Dakota will succeed in moving from a position of one of the most medically needy states in the nation to one in which the number of physicians is sufficient, and it will also help South Dakota acquire a distribution of specialties which is most proper. The ultimate result should be appropriate, accessible medical care for South Dakotans at a reasonable cost.

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- Educational programs relating to the hazards of **smoking** during pregnancy;
- Educational and promotional programs on the importance and proper use of **infant/child automobile safety restraints;**

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Last year the AMA Auxiliary "lost" nearly 19,000 members — These are grim statistics! The success of our endeavors, as well as that of future undertakings, can only be assured through dedicated membership. RENEW the present membership. RECRUIT the new member. REFRESH the non-member. REACTIVATE the drop-out member. STUDENT SPOUSE: REACH OUT to the resident physician and medical student spouse programs. THEY ARE OUR FUTURE!

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Mary Wrage

Mary Wrage, President
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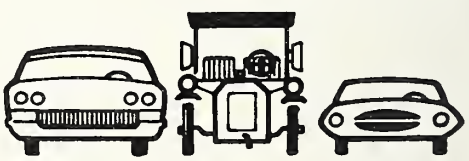

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Editor's Note:
Due to an error in printing, the x-rays in this article, which ran in the May issue, were inadvertently reversed. For the convenience of our readers, we are repeating the entire article.

Twenty Year Old Caucasian Male with Elevated Hematocrit and Right Lower Pole Renal Mass

Hoadley M. Harris, M.D.*
Richard A. Jaqua, M.D.†
Roger Nutt, M.D.‡
Discussers

John F. Barlow, M.D.§
Editor

Case #988 688

This 22 year old Caucasian male was admitted to Sioux Valley Hospital for evaluation of an elevated hematocrit and a right lower pole renal mass.

The patient had had bilateral testicular pain especially at night for two years, associated with painful erections. Ten months previously he had developed right flank and lower abdominal aching which was poorly localized and deep but with no definite radiation to the groin. There was no relationship to meals, or associated gastrointestinal or urinary symptoms. There was no hematuria. He was a nonsmoker. White blood count was 1.9 × 10^9/L, red blood count 8.02 × 10^{12}/L, hemoglobin 21 gm/dl, hematocrit 65 Vol/dl, mean corpuscular volume 81 fl, mean corpuscular hemoglobin 26 pg, mean corpuscular hemoglobin concentration 32%, red cell distribution width 14.7, mean platelet volume 8.5, platelet count 233 × 10^9/L, reticulocyte count 0.9%. A differential count showed 77% neutrophils, 1% neutrophilic bands, 19% lymphocytes, 2% monocytes, 1% eosinophils with 1+ anisocytosis, microcytosis, and poikilocytosis of the red cells on smear. A bone marrow examination showed erythroid hyperplasia. Arterial blood gas values, vitamin B-12 level, carboxyhemoglobin and hemoglobin electrophoresis were within normal limits. An erythropoietin level showed borderline elevation. A leukocyte alkaline phosphatase was depressed at 8. The patient was treated with multiple phlebotomies. The hematocrit dropped and the abdominal pain disappeared. The hematocrit again rose into the high 60's and a repeat erythropoietin level was markedly elevated. An intravenous pyelogram and computer tomographic scan (CT) showed a large, solitary, smooth cystic structure in the lower pole of the right kidney which was diagnosed as a possible simple cyst. He was referred for renal surgery. A hematology consult felt that the patient had secondary erythrocytosis.

A review of past medical history showed the patient had no other major symptoms. He had been evaluated at age 14 after an appendectomy because of short stature at which time he was 139 cm (4′ 1.5″) and 30 kgs (65 lbs). However, tests of the thyroid, adrenal and pituitary function were within normal limits. Additional tests included 12-panel chemistry which was normal except for an elevated alkaline phosphatase consistent with the patient's age. Skull and bone x-rays for age were unremarkable. Urinalysis was within normal limits and hematocrit was 41 Vol/dl with no other abnormalities on the complete blood count.

PHYSICAL EXAMINATION: Height 175 cm (5′ 2.5″); weight 48 kgs (106 lbs); pulse 80/min and regular; respirations 18/min and regular; blood pressure 120 systolic and 76 diastolic. The patient was alert, cooperative and in no distress. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The heart was not enlarged and had a normal rhythm with no murmurs or extra sounds. The abdomen was soft, flat and without tenderness, palpable organs or masses. The testes were descended. Rectal examination showed a smooth, nontender, small prostate. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis — yellow, clear; specific gravity 1.033; pH 6.5; negative for protein, glucose, ketones, bilirubin and hemoglobin; sediment negative. Hemoglobin 16.8 gm/dl, hematocrit 53 Vol/dl, mean corpuscular volume 76 fl, mean corpuscular hemoglobin 25 pg, mean corpuscular hemoglobin concentration 32%, total white count 7.2

* Resident in Family and Community Medicine, program affiliated with USD School of Medicine, Sioux Falls, SD.
† Professor and Chairman, Department of Laboratory Medicine, USD School of Medicine; Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD.
‡ Specialist in Internal Medicine; Clinical Faculty of Medicine, USD School of Medicine, Yankton, SD.
§ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

10^9/L with 48% segmented neutrophils, 45% lymphocytes, 2% eosinophils and 4% monocytes. The platelets were normal in morphology and the red cells showed slight anisocytosis and poikilocytosis on smear. The platelet count was 261 × 10^9/L. Lactic dehydrogenase (LD), alkaline phosphatase, asparate aminotransferase (AST, SGOT), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. An erythropoietin level was within normal limits. A chest film showed minimal calcific changes. An ultrasound-guided percutaneous puncture of the renal mass was attempted but no fluid could be aspirated. Internal echoes were seen within the cystic mass and an arteriogram was suggested. A right transfemoral selective renal arteriogram showed an avascular mass in the lower pole of the right kidney. An operation was performed.

DR. HARRIS: The signs and symptoms of renal neoplasms include hematuria, pain, palpable mass, hypertension, and manifestations of paraneoplastic syndromes.

Hematuria in renal neoplasms is more common in malignant than benign lesions and it may be gross or microscopic. Hematuria can also be seen in benign prostatic hyperplasia due to dilated bladder neck veins as well as in a variety of conditions involving the entire urinary tract.

Renal capsular distention is believed to be the cause of pain in the costovertebral angle region associated with renal neoplasms. Pain can also be caused by ureteral colic secondary to hyperperistalsis of the pelvis and ureter due to the passage of blood clots or fragments of tumor. Testicular pain is usually absent unless testicular torsion, bleeding, or infection are present. The painful erections in this case may be explained by erythrocytosis and the accompanying abnormal blood flow.

Palpable masses in the kidney include not only neoplasms but simple renal cysts, polycystic kidneys, and hydronephrosis. Pancreatic cysts or occasionaly splenomegaly can give palpable masses in the renal area.

An elevated blood pressure may be seen in up to 50% of the cases with Wilms' tumor but is rarely seen with renal cell carcinoma. Juxtaglomerular cell adenoma is a very rare renal neoplasm which secretes renin and produces hypertension in nearly all cases.

Paraneoplastic syndromes associated with renal tumors fall into four distinct areas. The first includes systemic manifestations manifested by fever seen in about 20% of renal cell carcinoma cases and secondary amyloidosis which can lead to renal failure and hepatosplenomegaly. The second encompasses hematologic manifestations. These include anemia seen in 35% of the renal cell carcinomas and erythrocytosis seen in 4% of renal tumors. Benign as well as malignant conditions of the kidney can be associated with an elevated hematocrit. In addition, an elevated erythrocyte sedimentation rate may be present. Thirdly, the gastrointestinal syndrome may present with abnormal liver function tests in the absence of hepatic metastases including abnormal prothrombin time, bilirubin, and alkaline phosphatase values which revert to normal after nephrectomy. This syndrome has been reported in up to 10-15% of the cases in some series. Lastly, endocrine manifestations have been seen. These include hypercalcemia secondary to metastatic bone disease or secondary to excretion of parathyroid hormone-like or prostaglandin-like substances from the kidney in the absence of metastases. Rarely ectopic secretion of adrenocorticotropin (ACTH) can produce Cushing's syndrome. Even rarer is gynecomastia secondary to secretion of gonadotropins by renal tumors.

I will now discuss some of the more common renal tumors. The only malignancy which tends to metastasize to the kidney is a neoplasm of the other kidney. Therefore, when we talk about renal neoplasms we are usually discussing primary tumors. Among the benign tumors is a cortical adenoma, a lesion derived from the renal tubular epithelium, which is seen in 7-22% of autopsies and is usually located in the cortex. By definition, it is less than 3 cm in size and it is composed of cells similar to those seen in renal cell carcinoma. Some feel this is simply a small renal cell carcinoma.

An oxyphil adenoma or oncocytoma of the kidney is a benign lesion which can reach large size and has a characteristic cell with abundant red cytoplasm filled with mitochrondria on electron microscopy.

The fibroma is composed of well differentiated fibroblasts and is a small lesion usually found in the renal pyramids at autopsy. It has no malignant potential.

The so-called renal hamartoma or angiomyolipoma is commonly seen in tuberous sclerosis, however, it may be seen in the absence of that disease and is composed of vessels, smooth muscle and fat. It is a benign lesion but may undergo spontaneous rupture or hemorrhage and cause pain and anemia. The lesions can often be diagnosed by angiography which shows multiple small aneurysms.

Hemangiomas may be composed of blood vessels or lymph vessels and are benign congenital lesions which usually occur in the medulla of the kidney. Their most common site is the liver.

Malignant tumors — Renal cell carcinoma (Grawitz tumor or hypernephroma) was described in 1883 and was named hypernephroma because it appeared similar on gross appearance to the adrenal cortex. These lesions constitute 85% of the primary malignant renal tumors. It is totally unpredictable. The primary may be silent and the presenting symptoms may be due to metastases to the brain, bone or lung. These metastases may be single and some have

suggested that if they are removed along with the primary, reasonable survival can be expected in some cases. The renal primary may grow to large size and be silent without metastases. On the other hand, growth may be explosive. Therefore, prediction of survival is very difficult. The microscopic morphology of these tumors is also highly variable. As mentioned, cortical lesions below 3 cm in diameter with typical well differentiated histology are classified as cortical adenomas by some authors. Renal cell carcinomas usually appear after the fourth decade, and often in the upper poles of the kidney as solitary lesions. They are spherical masses 3-15 cm in diameter and are bright yellow or gray. They show pseudoencapsulation but are not truly encapsulated. Over 50% will invade the renal vein or grow in a column of tumor into the inferior vena cava. Invasion of these vascular structures as well as invasion of the renal capsule and surrounding perinephric region are considered poor prognostic features. The tumor characteristically is very vascular, a feature helpful in angiographic diagnosis. There are a variety of histologic patterns including papillary, tubular, glandular, and undifferentiated forms such as sarcomatoid varieties. The clear cell carcinoma variant is the most common and has many of the ultrastructural features of tubular epithelium. This clear cell pattern can often help the pathologist in recognizing the primary when a metastasis is removed from a distant organ. Since there is abundant lipid in the cells, the presence of fat in aspirates of renal masses may be sign of malignancy.

I will now return to some of the classic clinical aspects. Hematuria is seen in 60-90% of the cases but pain and a palpable abdominal mass are usually late symptoms. It should be pointed out that the classic triad of hematuria, pain and palpable abdominal mass is only seen in 9% of the cases of renal cell carcinoma. Hematuria in conjunction with the paraneoplastic syndromes, accounts for most presentations of renal cell carcinoma. A high index of suspicion followed by radiologic diagnosis in renal cell carcinoma is a must because of the wide variety of symptoms. These tumors metastasize hematogenously to the lungs (50%) bones (33%) and to the opposite kidney (15%) as well as to lymph nodes, liver, brain and adrenals. Because the tumor exhibits bizarre growth patterns and may be slow growing and silent or grow in explosive nature as well as having a variety of histologic patterns, it is a most confusing tumor.

Treatment is also perplexing. A renal neoplasm thought to be cured by surgery may develop metastasis 10-20 years later. The overall 5 year survival is 35%. This rises to 50% if it is confined to the kidney (stage I). The prognosis drops to the 10% range if the renal vein or perinephric fat are involved. Chemotherapy and radiation have been only slightly successful in treating this neoplasm. The major hope of cure is extirpative surgery at an early stage.

The second most common malignant tumor of the kidney is the Wilms' tumor (embryoma or nephroblastoma). This tumor is second only to neuroblastoma as a solid malignant neoplasm in viscera in children. Sixty-five percent of cases occur in children under 3 years of age. The tumor contains a variety of cellular tissues so that some believe it is a form of teratoma. The tumor is usually a large expansile unilateral mass which can produce abdominal distention with a palpable mass. In fact, most children present with an abdominal mass while hematuria and abdominal pain are less common. The tumor may cause intestinal obstruction and pulmonary metastases are not uncommon at the time of tumor diagnosis. Fortunately, with a combination of chemotherapy, radiation and surgery, a two year survival of over 90% even with metastases to the lungs has been achieved. When metastases occur to other organs such as the bone, brain and liver, the survival is less.

What are some of the diagnostic modalities used for diagnosis of renal tumors? In the abdominal plain film an enlarged kidney with bulging contour and obliteration of the psoas shadow are indications of renal neoplasm. The psoas shadow may be seen through a cyst but not a solid tumor.

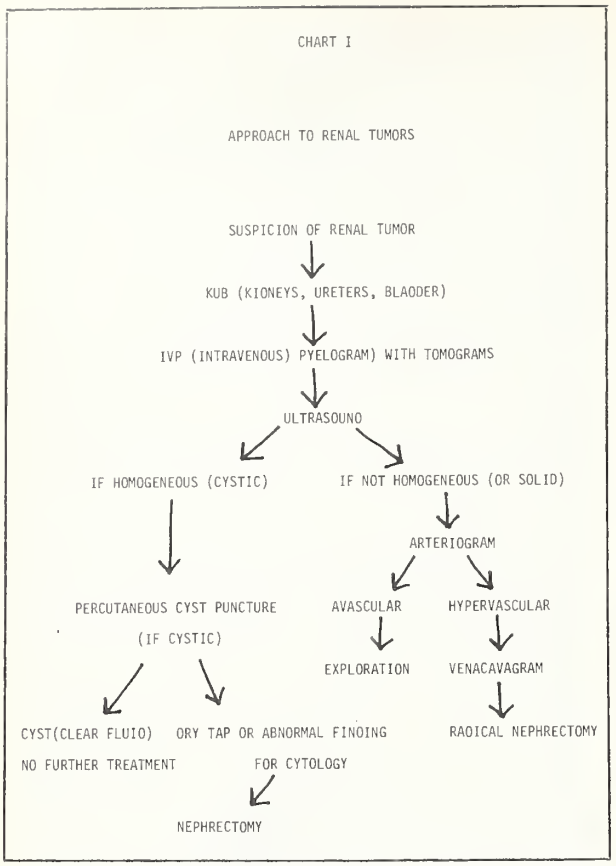
In the intravenous pyelogram, a filling defect or distortion of the calyceal pattern may occur. Very rarely a renal cell carcinoma may compress the ureter and produce secondary hydronephrosis. If renal vein involvement occurs, there may not be excretion of radiocontrast material by the kidney. This is a bad prognostic sign. Tomograms of the kidney may show the tumor to better advantage.

The sonogram is very helpful in diagnosis of renal tumors because it can distinguish cysts from solid neoplasms in 95% of the cases. If there is any question, a percutaneous needle puncture may be obtained. If clear fluid is obtained, the lesion is a cyst. If bloody fluid is obtained, a tumor may be present and cytologic examination of the fluid should be performed. Biopsy of renal neoplasms can also be carried out under sonographic guidance.

There is some question whether the computer tomogram is of any additional help in differentiating cyst from solid tumor in the kidney.

However, angiography is of considerable value because of the characteristic neovascularity in renal cell carcinoma. Pooling may also occur in the tumor but not in the cyst.

Finally, I would like to outline an overall approach to the diagnosis of renal tumors (Chart I). First, one must be very suspicious of a renal tumor.



A simple x-ray of the kidneys, ureter and bladder (KUB) may show a mass or an enlarged kidney with an irregular outline. An intravenous pyelogram (IVP) with tomograms is the next step. A sonogram to distinguish between a cystic or solid mass can then be performed.

Cystic lesions can be aspirated and cytology performed if fluid is bloody. No further treatment is usually necessary although the wall can be destroyed by sclerosing solutions. If the tap is dry or abnormal cells are present on cytologic examination, nephrectomy is indicated. If the lesion is not homogeneous by sonogram, an angiogram is necessary. If a typical hypervascular pattern is seen, a radical nephrectomy perhaps preceded by an inferior venacavagram is necessary. Exploration is also indicated for avascular neoplasms as in this case.

The tumor we had today was in the wrong age group for a renal cell carcinoma and was avascular on the angiogram. I think from the studies I can safely say it was a neoplasm and not a cyst. Other than that, I can not be more specific.

Dr. Harris's Diagnosis
Neoplasm of Kidney

DR. BARLOW. Thank you for your excellent discussion. I agree this is a most unusual neoplasm and would be impossible to diagnose without pathologic examination.

Submitted was a 140 gm 11 × 5.5 × 3.5 cm right kidney. In the lower pole was a 4 cm in greatest dimension smooth tan homogeneous well demarcated mass which bulged from the surface but did not invade the perirenal fat (Fig. 1). Microscopically the lesion was well demarcated and in some areas a pseudocapsule was seen. There was no invasion of the surrounding parenchyma. Most of the tumor was composed of masses of tubules lined by cells with scanty cytoplasm but uniform round to oval nuclei without hyperchromatism. Mitoses were rare. In other areas a marked papillary arrangement with psammoma bodies was present (Fig. 2). Structures consisted with glomerulotubular structures but probably papillae were seen in solid areas of the tumor. Necrosis, macrophage infiltration or collections of foam cells were not seen.

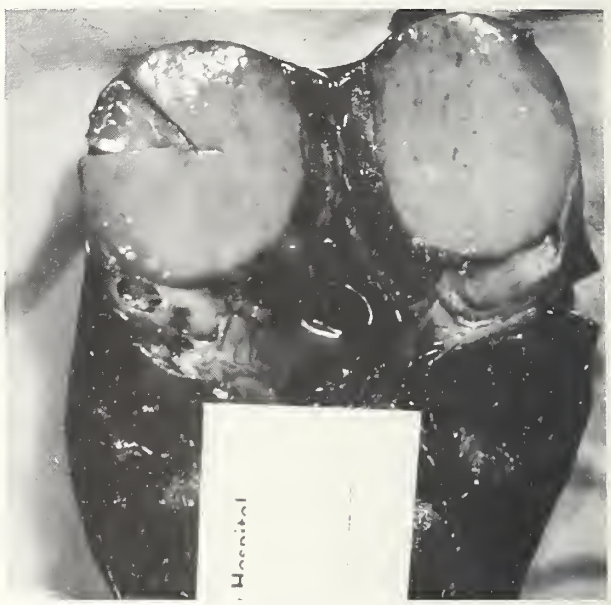


Figure 1
Gross picture of cut section of solid well circumscribed renal tumor.

One thought was that this might represent a Wilms' tumor in an adult. However, the frank papillary areas and psammoma bodies made this unlikely. In addition, the tumor was sent to Dr. George Fairow of the Mayo Clinic, Department of Surgical Pathology and he felt the lesion was a papillary carcinoma.

I think it is of interest that 167 cases of adult Wilms' tumor have been reported in adults. Some of these may represent sarcomatoid variants of renal carcinoma and some authors may have confused papillary arrangements with glomerulotubular structures characteristic of Wilms' tumor (Fig. 3).

DR. JAQUA: When the light microscopy of this tumor was presented at our noon surgical conference there were two questions which I felt ultrastructure might help answer. The first would be to look for

mesenchymal or glomerular type differentiation to help differentiate Wilms' tumor from a papillary adenocarcinoma, and secondly to see if we could demonstrate any ultrastructural evidence for secretory activity in the tumor to correlate with the elevated erythropoietin levels.

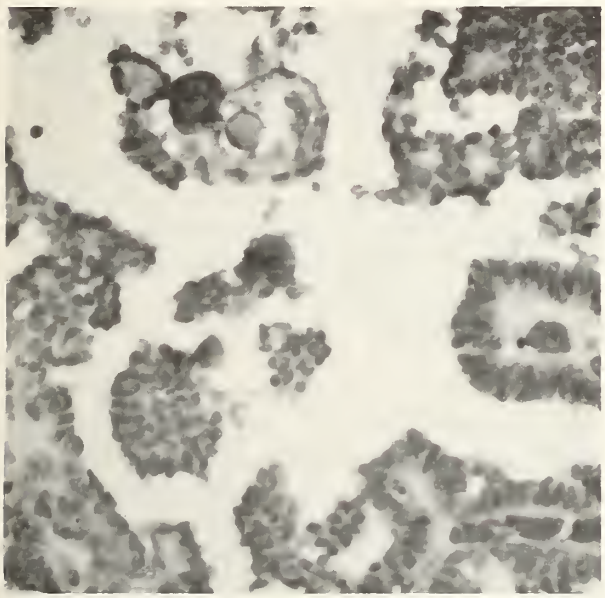


Figure 2
Papillary features with psammoma bodies (upper center).

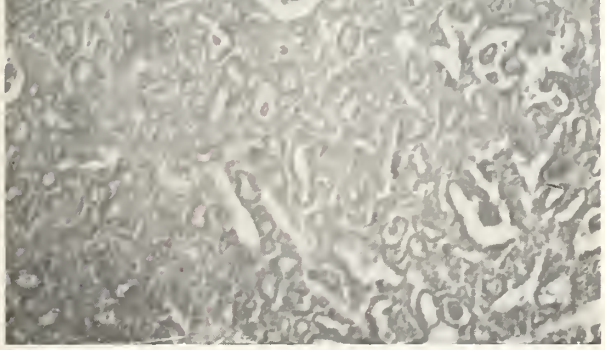


Figure 3
Pseudoglomeruloid structures suggestive of Wilms' tumor.

We examined numerous blocks from this tumor by both thick and thin section morphology and were unable to demonstrate any mesenchymal or glomerular type differentiation. The tumor was nearly entirely made up of tubular-like structures with well developed basal lamina, a small, often collapsed lumen and with polarized columnar cells exhibiting well developed junctional complexes at their apices along with true microvilli and an occasional cilium extending into the lumen (Fig. 4). No secretory activity was identified in the epithelial cells or in stromal cells. Unlike renin, erythropoietin has not been localized to an ultrastructurally defined granule, and although studies are conflicting, the immunochemical studies which have been done seem to point to the glomerulus as the potential renal source. Since this tumor tissue was not assayed for renin, the possibilities of tumor compression of normal renal parenchyma and secondary prostaglandin stimulation of erythropoietin must also be considered in this case. I believe the ultrastructure confirms the renal epithelial origin of this neoplasm.

Figure 4
Electron micrograph showing cells in a tubular arrangement favoring renal tubular origin of this tumor.

DR. BARLOW: Papillary carcinomas are rare tumors. One series reported 34 cases culled from 224 renal cell carcinomas. The majority of papillary carcinomas (85%) were confined to the kidney (stage I) compared to only half of the nonpapillary varieties. Papillary carcinomas had a better survival rate than other renal cell carcinomas even though massive necrosis, a poor prognostic feature of nonpapillary carcinomas, was frequently seen in the papillary renal neoplasms. Psammoma bodies and

calcification identified on x-ray was common. An interesting finding was atypical proliferation of the collecting tubules near the tumor suggesting an origin from these structures. This was not noted in this case. It must be stressed, however, that these tumors can invade the renal vein and produce multiple metastases, but lesions which did so did not show features considered to be associated with a good prognosis — encapsulation, necrosis or macrophage infiltration. The lesion today can probably be considered encapsulated but did not show macrophage infiltration or necrosis. The fact that the lesion was confined to the kidney (stage I) would indicate a good prognosis.

Papillary tumors characteristically are hypovascular or avascular on arteriography, a finding nicely demonstrated by this case (Fig. 5).

Figure 5
Arteriogram of kidney with absent vascularity in lower pole of kidney of this case typical of papillary carcinoma.

Although papillary carcinomas of the kidney have been associated with hypertension and hypercalcemia, I did not see mention of a case with erythropoietin production and secondary erythrocytosis. Secondary erythrocytosis has been described in over 350 neoplasms. Over half or 179 have been from the kidney. One hundred twenty of these were hypernephromas. Wilms' tumor, sarcomas, and renal cysts have also been known to secrete erythropoietin. However, only 1-10% of all renal cell carcinomas cause secondary erythrocytosis and only 1% of cases of erythrocytosis are due to renal cell carcinoma. There was a study showing that 63% of cases of renal cell carcinoma secrete erythropoietin but this does not usually result in an elevated hematocrit. This latter finding has been associated with an improved prognosis.

FINAL ANATOMIC DIAGNOSIS
PAPILLARY CARCINOMA OF THE RIGHT KIDNEY

*DR. KEITH A. ANDERSON: Would you clarify that point on the presence of fat in cytologic preparations from aspiration of renal lesions?

DR. HARRIS: As I understand it, there seems to be a high correlation between the presence of high lipid levels in fluid aspirate and the presence of malignancy.

DR. BARLOW: I would like to introduce Dr. Roger Nutt, the physician who referred this case to Dr. John Sall who subsequently operated on this patient.

DR. NUTT: I can provide some followup. The patient is doing extremely well and now has an hematocrit of 42% several months after surgery.

†DR. TOM GAECKLE: What do you feel is the mechanism for erythropoietin secretion in this case? Isn't it true that erythropoietin secretion occurs less commonly in cysts than tumors of the kidney?

DR. BARLOW: I am sure it depends in what series you read, but if one were to take autopsy series, I am sure erythropoietin secretion by renal cysts would be considerably less than 1%.

DR. JAQUA: I think one has to realize that some feel that it is the compression of the surrounding kidney which is responsible for erythropoietin or renin secretion by the kidney in the case of any expansile condition such as renal cyst or renal tumor. This may be mediated by prostaglandin elevation which in turn stimulates increased erythropoietin levels. Juxtaglomerular tumors have been described that actually secrete prostaglandins and elevate erythropoietin in this fashion.

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* Resident in Pathology, Sioux Valley Hospital and Veteran's Administration Hospital, Sioux Falls, Program Affiliated with School of Medicine, University of South Dakota, Sioux Falls, SD.

† Specialist in Internal Medicine, Sioux Valley Hospital, Clinical Faculty of USD School of Medicine, Sioux Falls, SD.

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Ninth Annual Convention of the American College of International Physicians, Marriott Hotel, Chicago, IL, Aug. 15-19. AMA Category I credits available. Contact: ACIP, 3030 Lake Ave., Fort Wayne, IN 46805. Phone: (219) 424-7414.

Geriatric Medicine Program for Faculty of Family Practice Residencies, Minneapolis Plaza Hotel, Minneapolis, MN, Aug. 22-24. Fee: $200. 17.5 hrs. AAFP & AMA Category I credits. Contact: Off. of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

September

Radiology/84: Thoracic Imaging, Willey Hall, U. of Minn., Minneapolis, MN, Sept. 10-14. Fee: $450. 28 hrs. AMA Category I credits. Contact: Off. of CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Ninth Annual Perinatal Conference, Holiday Inn of the Northern Black Hills, Spearfish, SD, Sept. 13-14. Contact: Judie Prince Crane, R.N., S.D. Perinatal Asso., Box 5039, Sioux Falls, SD 57117-5039. Phone: (605) 333-7193.

Management of Alzeheimer's Disease, Willey Hall, U. of Minn., Minneapolis, MN, Sept. 17-19. AMA Category I credits available. Contact: Off. of CME, Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Trauma & Critical Care Seminar, Pillsbury Aud., Hennepin County Med. Ctr., Minneapolis, MN, Sept. 20-22. AMA Category I credits available. Contact: Off. of CME, Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Request Topics for Clinicians, Marriott Hotel, French Quarter, New Orleans, LA, Sept. 21-23. 11 hrs. AMA Category I credits. Fee: $225. Contact: Dennis Occhipinti, M.D., Prog. Cood., 5640 Read Blvd., #400, New Orleans, LA 70127.

Coronary Heart Disease Workshop, Wilder Forest, Stillwater, MN, Sept. 23-25. AMA Category I credits available. Contact: Off. of CME, Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Aging, Vitality & Running, Amfac Hotel, Minneapolis, MN, Sept. 28-29. AMA Category I credits available. Contact: Off. of CME, Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone (612) 373-8012.

October

Research Day, Battenfeld Aud., U. of Kans. Med. Ctr., Kansas City, KS, Oct. 5. 7 hrs. AMA Category I credits. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

American College of Chest Physicians' 50th Annual Scientific Assembly, Loews Anatole Hotel, Dallas, TX, Oct. 8-12. 30 hrs. AMA Category I credits. Contact: ACCP, 911 Busse Highway, Park Ridge, IL 60068-2375. Phone: (312) 698-2200.

International Symposium on Human Nutrition & Development, Washington, DC, Oct. 17-21. Fee: $350. 22 hrs. credit. Contact: Symposia Medicus, 2880 Shadelands Dr., #404, Walnut Creek, CA 94598. Phone: (415) 935-7889.

39th Annual Internal Medicine Symposium, Battenfield Aud., U. of Kans. Med. Ctr., Kansas City, KS, Oct. 18-19. AMA & AAFP credits available. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

52nd Annual Postgraduate Assembly; Omaha Mid-West Clinical Society, Red Lion Inn, Omaha, NE, Oct. 29-31. Contact: Lorraine Seibel, Exec. Sec., Omaha Mid-West Clinical Soc., 7363 Pacific St., #210-A, Omaha, NE 68114. Phone: (402) 397-1443.

November

Annual Diabetes Symposium, Battenfeld Aud., U. of Kans. Med. Ctr., Kansas City, KS, Nov. 2. AMA & AAFP credits available. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

Primary Care of the Child with a Developmental Disorder, Gillette Children's Hosp., St. Paul, MN, Nov. 2-3. AMA credits available. Contact: R. Brett Day, Ed.S., CME Dept., Gillett Children's Hosp., 200 E. University Ave., St. Paul, MN 55101. Phone: (612) 291-2848.

Lung Cancer 1984, Shamrock Hilton Hotel, Houston, TX, Nov. 7-9. Contact: Off. of Conf. Services, Box 131, M. D. Anderson Hosp. & Tumor Instit., 6723 Bertner Ave., Houston, TX 77030. Phone: (713) 792-2222.

89th Annual Meeting of the American Academy of Ophthalmology, Georgia World Cong. Ctr., Atlanta, GA, Nov. 11-15. Contact: Am. Acad. of Oph., Box 7424, San Francisco, CA 94120-7424.

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Published Monthly by the S.D. State Medical Assn.

Volume XXXVII August 1984 Number 8

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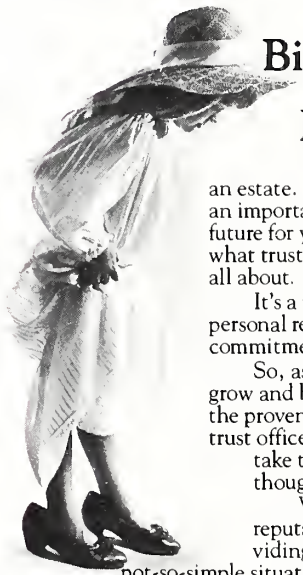

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Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

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J-4044 January 1984

SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 8 August 1984 (ISSN 0038-3317)

Official Publication of the
South Dakota State
Medical Association

the South Dakota Chapter
of The American Academy
of Family Physicians

the South Dakota Chapter
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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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NEXT MONTH

Use of the Epidural Blood Patch in the Treatment of the Post Dural Puncture Headache

Clinicopathological Conference
Twenty-Seven Year Old Postpartum Gravida IV Para IV With Hypotension and Chest Pain

HEALTH SCIENCES LIBRARY

The ''old AMA'' and the ''new AMA'' are a marked contrast in style. The ''new AMA'' shows an amazing growth and grasps the problems of today in medicine and delivery of health care. Thirty-five years ago the problems and challenges of that time were being met. Today the new challenges and federal impacts produce a totally new set of problems. Most of us, I am sure, peruse the JAMA and AMERICAN MEDICAL NEWS which give us the current situation. Having just returned from the annual AMA meeting, I must say this is the most democratic organization I have seen. The addition of the resident and student sections also give everyone a new perspective. These young people have done their homework and their presentations were excellent. The House of Delegates, Board and officers are our best representation in promoting medicine today.

Your South Dakota Medical Association will continue to promote and protect your interests through our AMA delegates and state officers. I would hope any of you who are not AMA members will give serious consideration to becoming a member. The AMA has been called names, both good and bad, but the AMA is the only real organization with clout to protect medicine in the changing world of today so we may continue to give our patients (John Q. Public) the best medical care in the world.

Howard L. Saylor, Jr., M.D., President
South Dakota State Medical Association

Transactions Of The South Dakota State Medical Association 103rd Annual Meeting June 7, 8, 9, 10, 1984

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REPORT OF THE BUDGET AND AUDIT COMMITTEE

Thursday, 8:30 a.m. **Howard Johnson Motor Lodge**
June 7, 1984 **Rapid City, South Dakota**

The meeting was called to order at 8:30 a.m. by Jay Hubner, M.D., Chairman of the Committee. Present for roll call were Drs. Hubner, Joseph Hamm, Howard Saylor, Richard Gere, W. O. Rossing, Robert Ferrell, Frank Messner, G. E. Tracy, and Robert Johnson, Executive Secretary.

The Committee reviewed the CPA Audit prepared by McGladrey, Hendrickson & Pullen. Mr. Johnson discussed several items of particular interest to the Budget and Audit Committee. Dr. Saylor moved that the Committee accept the CPA Audit. The motion was seconded and carried.

Mr. Johnson discussed a tentative meeting with Governor Janklow to discuss cost containment proposals. It was determined that a meeting could be arranged the week of July 16, if it is convenient for the Governor and his staff.

The meeting adjourned at 9:30 a.m.

FIRST COUNCIL MEETING MINUTES

Thursday **Lincoln Room**
June 7, 1984 **Howard Johnson Motor Lodge**
10:00 a.m. **Rapid City, South Dakota**

The meeting was called to order by Robert Ferrell, M.D., Chairman of the Council at 10:00 a.m. Those members present were Drs. Joseph Hamm, J. A. Eckrich, Jr., Howard Saylor, James Larson, Richard Gere, R. C. Jahraus, W. O. Rossing, David Buchanan, Durward Lang, Charles Monson, Robert Ferrell, Michael Pekas, G. E. Tracy, R. E. Gunnarson, Frank

Messner, Bruce Lushbough, G. H. Steele, A. A. Lampert, Jr., Curtis Wait, Lowell Hyland, Jay Hubner, R. I. Porter, James Jackson, Ed James, M. George Thompson, Joseph Kass, D. G. Ortmeier, Gene Koob, Thomas L. Krafka, James Reynolds, and Morris L. Radack.

Dr. R. H. Quinn from the School of Medicine, Dr. John Gregg from the State Health Department, Dr. Nathaniel R. Whitney, and Dr. Dennis Johnson were also present.

Dr. Ferrell introduced Steve Ellwing, South Dakota's field representative from the American Medical Association.

Dr. Saylor moved to accept the minutes of the previous meeting as printed and distributed. The motion was seconded and carried.

I. MEDICARE CODES AND LANGUAGE — Dr. Saylor reported on Medicare codes. **He moved that an appropriate letter of reprimand be forwarded to North Dokato Blue Shield, the Denver office, the Baltimore office, and also to our Congressional Delegation. The motion was seconded and carried.**

II. USDSM ADMISSIONS COMMITTEE — Dr. Ferrell provided information for the Council members. He stated that all students are selected on the basis of the same criteria and without any special considerations. This was accepted for information only.

III. MEDICAL SCHOOL UPDATE — An update on the Medical School was given by Dr. Quinn and handouts were distributed. He reported that the class size has been cut to 50 students this year. He also reported on budget, space, Indian Health, and several other areas of concern to the school. This was accepted for information.

IV. REPORT OF CME ACCREDITATION COMMITTEE — Dr. Koob briefed those present on the Continuing Medical Education Accreditation Committee.

MINUTES OF THE CONTINUING MEDICAL EDUCATION ACCREDITATION COMMITTEE

3:00 p.m. **Airport Holiday Inn**
Friday, April 13, 1984 **Sioux Falls, South Dakota**

The meeting was called to order by K. Gene Koob, M.D., Chairman. Those present for roll call were Doctors Koob, James Larson, Robert Talley and Mark Harlow.

I. REVIEW OF FEE SCHEDULE FROM ACCME. The ACCME committee will review the State Medical Association in 1985 for accreditation purposes provided a $250 fee is remitted. **Dr. Larson moved that the State Medical Association remit the $250 fee and request this review. The motion was seconded and carried.**

II. REVISED ESSENTIALS AND GUIDELINES. The revised essentials and guidelines proposed by ACCME were reviewed by the committee. **Dr. Larson moved that the Committee accept the revised essentials and guidelines as proposed. The motion was seconded and carried.**

III. PROCEDURAL AND ADMINISTRATIVE INFORMATION ON ACCREDITATION. The Committee reviewed this information and noted that the address on the form should be changed from the national office address to the South Dakota State Medical Association name and address. This was accepted for information only.

IV. REVISED APPLICATION FORM FOR SPONSOR ACCREDITATION. The Committee noted this is the form the individual organization completes about its program. This was accepted for information only.

V. REPORT OF ACCME CONFERENCE FOR STATE MEDICAL SOCIETIES. It was noted that the South Dakota Medical Association has not established an appeals process as ACCME requires. **Dr. Harlow moved that the following appeals process be established: 1) If the CME committee denies a request for accreditation, the applying organization or institution can request a repeat review in front of the CME committee. 2) If this committee does not approve the application, the applicant can request a review before the Council of the State Medical Association. The motion was seconded and carried.** The committee then discussed procedures to handle complaints for improperly awarded credits. **Dr. Talley moved that the following procedure be established to handle complaints for improperly awarded credits: 1) First a complaint must be received by the committee. 2) The committee chairman would designate a member of the committee to investigate the complaint and report back to the committee. 3) If the organization or institution is found in noncompliance, remedial action will be taken as deemed necessary by the committee. The motion was seconded and carried.** The Committee then discussed ways to monitor accredited organizations or institutions. **Dr. Harlow moved that the committee ask each accredited organization or institution to submit a list of programs they will sponsor during the year, the committee chairman will assign a member of the committee to attend at least one program of each accredited organization, that committee member will report back to the committee and if any variance is noted the committee will take whatever action it deems necessary. The motion was seconded and carried. Dr. Larson moved that the Committee notify ACCME that the Report of the ACCME Conference is acceptable. The motion was seconded and carried.**

VI. APPLICATION FOR REACCREDITATION. This was a letter used to notify accredited organizations or institutions of the need for resurvey and was received for information only.

VII. REPORT OF SITE SURVEY TEAM. The committee reviewed the report of the survey on the Black Hills Seminar which is sponsored by the South Dakota Chapter, American Academy of Family Physicians. The committee directed the executive office to correspond with ACCME regarding the definition of interstate and intrastate for purposes of accrediting organizations. **Dr. Larson moved that the committee grant approval to the South Dakota Chapter, American Academy of Family Physicians provided ACCME's definition of interstate and intrastate and their requirements are compatible with this approval. The motion was seconded and carried.**

VIII. SDSMA SURVEY ON CME. Lorin Pankratz briefly reviewed information on the SDSMA survey and the responses concerning CME requirements for licensure and for membership in the State Association. This was for information only.

The meeting adjourned at 4:30 p.m.

Dr. Gere moved to approve the report as presented. The motion was seconded and carried.

V. CME MEMBER AUDIT — The Council received the report on the random audit of SDSMA members regarding CME hours and accepted it for information only.

VI. CHELATION THERAPY — Mr. Johnson briefly explained the response from the Board of Medical Examiners regarding chelation therapy. The Board will not expand in anymore detail until the study is completed. **Dr. Rossing moved that the Council draft a statement for the Grab Bag and/or Journal based on the AMA's recommendation that chelation therapy is not acceptable treatment for coronary or other arterial atherosclerosis. The motion was seconded. Dr. Pekas amended this motion to include information on when EDTA chelation therapy is acceptable and appropriate. The amendment was seconded and carried. The motion as amended was carried.**

VII. HEALTH DEPARTMENT UPDATE — Dr. John Gregg from the State Health Department gave an update on that organization. He reported on the following items:

1. The position of the Department regarding a statewide tumor registry. It was requested that the council act on this item.
2. Computerization of the craniofacial anomalies patients, and the development of this as a service, and a research project.

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3. The Fetal Alcohol Syndrome project of the Department. Special Project Status.
4. The dermatographic study now in progress to find a genetic marker to identify people prone to procreate offspring with craniofacial, cardiac, and renal anomalies.
5. The Primary Care Project for South Dakota.
6. Medical School liaison. Indian Health. Committees.
7. The Office of Medical Services of the Department in the future.

Dr. Tracy moved that the Council refer item #1 to the Commission on Scientific Medicine. The motion was seconded and carried.

VIII. REQUEST FOR CONTRIBUTION — Mr. Johnson reported that he had received a request for $300 for the Midwestern Conference of the Council of State Governments to assist in hosting their annual meeting. **Dr. Saylor moved that the SDSMA donate up to $50 at the discretion of the Executive Secretary. The motion was seconded and carried.**

IX. PUBLIC RELATIONS PROGRAM — Mr. Lorin Pankratz discussed the outline provided to the Council for a Public Relations Program. This will be referred to the Commission on Internal Affairs Communications & Liaison for further study and development of a more comprehensive proposal for the Council's consideration at a later date.

The meeting adjourned at 11:30 a.m.

SECOND COUNCIL MEETING MINUTES

10:45 a.m. Lincoln Room, Howard Johnsons
Sunday, June 10, 1984 Rapid City, South Dakota

The meeting was called to order by Dr. Robert Ferrell, Chairman of the Council. Those present for roll call were Drs. Howard Saylor, Curtis Wait, Richard Gere, David Buchanan, W. O. Rossing, C. D. Monson, Joseph Hamm, K. Gene Koob, Robert Ferrell, R. E. Gunnarson, Frank Messner, Lowell Hyland, G. E. Tracy, Richard Porter, Bruce Lushbough, Jay Hubner, G. H. Steele, James Jackson, J. A. Eckrich, Jr., Thomas Krafka, James Larson, and Joseph Kass.

Dr. Lushbough moved to dispense with the reading of the minutes of the previous meeting inasmuch as they will be published. The motion was seconded and carried.

I. INTRODUCTION OF NEW COUNCILORS AND ALTERNATE COUNCILORS. Dr. Ferrell introduced Councilors, Dr. Curtis Wait, Brookings-Madison District; Dr. Jay Hubner, Yankton District; and Alternate Councilor, Dr. Thomas Krafka, Black Hills District.

II. ELECTION OF COUNCIL CHAIRMAN. Dr. Rossing nominated Dr. Robert Ferrell. **Dr. Saylor moved that nominations cease and a unanimous ballot be cast for Dr. Ferrell. The motion was seconded and carried.**

III. ELECTION OF SECRETARY-TREASURER. Dr. Messner nominated Dr. George Thompson to complete the unexpired term of Dr. Rossing as secretary-treasurer. **Dr. Saylor moved that nominations cease and a unanimous ballot be cast for Dr. Thompson. The motion was seconded and carried.**

IV. DATE FOR FALL COUNCIL MEETING. The Council determined that the next Council meeting would be held on September 14.

V. INFORMED CONSENT FORM. Dr. James Larson discussed the various forms now being utilized by physicians in South Dakota. **He moved that the State Medical Association develop a uniform form for informed consent for use by South Dakota physicians. The motion was seconded and carried.** Dr. Ferrell referred this to the Commission on Professional Liability for consideration and development of a form for the Council's review at a later date.

VI. RESOLUTION FROM YANKTON DISTRICT REGARDING SUPPORT FOR THE SOUTH DAKOTA HUMAN SERVICES CENTER. The House of Delegates referred this to the Council for review and revision. **Dr. Tracy moved to refer this to the appropriate commission asking them to review this resolution, making appropriate revisions and returning it to the Council for action at a later date. The motion was seconded and carried.** (One dissenting vote.)

VII. HOSPITAL BOARD BYLAWS. Mr. Johnson discussed a problem which has been brought to his attention regarding a small town hospital board and a possible conflict with the board's bylaws. **Dr. Buchanan moved to refer this matter to the Executive Commission when additional information is available and if a problem does, in fact, exist. The motion was seconded and carried.**

VIII. LOCUM TENENS PROGRAM FOR SOUTH DAKOTA. Mr. Johnson discussed a request which he received from a rural South Dakota physician for a locum tenens program. Following discussion **Dr. Hamm moved that the Council ask the appropriate commission to undertake a continuing and in depth study of the need for a locum tenens program in South Dakota and prepare a proposal for the Council's consideration at an upcoming meeting. The motion was seconded and carried.**

IX. PHYSICIANS ASSISTING THE BOARD OF MEDICAL AND OSTEOPATHIC EXAMINERS. Mr. Johnson reported that a physician recently assisted the Board of Medical and Osteopathic Examiners and inasmuch as Board records are open for public review this physician may now be asked to testify against a colleague in a malpractice case. **Dr. Saylor moved that the Association provide assistance to this physician and any other physician in a similar situation should South Dakota law require that this physician, in fact, testify in a malpractice case. The motion was seconded and carried.**

X. DISTRICT MEDICAL SOCIETIES. Dr. Hamm discussed his presidential visits to the twelve district medical societies during the past year. He encouraged councilors to report State Association activities to their members on a regular basis and keep their membership informed. It was noted that the executive office is preparing information which will be provided to new councilors and commission chairmen and members to assist them in their duties. This was accepted for information only.

XI. IMPAIRED PHYSICIANS COMMITTEE. Dr. Ferrell encouraged the Council and the State Medical Association members to take a greater interest in the Impaired Physicians Committee.

Dr. Saylor encouraged officers and councilors to express their opinions to him and he offered his assistance whenever possible during his year as president.

The meeting adjourned at 11:15 a.m.

MINUTES OF THE FIRST HOUSE OF DELEGATES MEETING

8:30 a.m. Howard Johnsons Motor Lodge
Friday, June 8, 1984 Rapid City, South Dakota

The meeting was called to order at 8:45 a.m. by Frank Messner, M.D., Speaker of the House. Mr. Robert Johnson, executive secretary took the roll. Present were the following members: Drs. Joseph Hamm, Howard Saylor, Jr., Richard Gere, William O. Rossing, Frank Messner, Gerald E. Tracy, Bruce Lushbough, Durward Lang, Jerome A. Eckrich, Jr., James Larson, Curtis Wait, R. Curtis Jahraus, David Buchanan, Charles D. Monson, Michael Pekas, Denny Ortmeier, Richard Gunnarson, Lowell Hyland, Richard Porter, Jay Hubner, Tom Krafka, Robert L. Ferrell, James Jackson, M. George Thompson, Joseph Kass, William R. Taylor, Winston Odland, Parry Nelson, W. Nicol Guddal, Richard Holm, Tad Jacobs, Ravi Kapur, William Huet, Charles Flohr, Gary Van Ert, James Ryan, A. J. Javurek, George Bruins, Dorrance Ensburg, Jerry Simmons, James Quale, David Hoversten, Bradley Randall, Rodney Parry, Jeffrey B. Hagen, Jerome W. Freeman, Guy Tam, Morris Radack, Larry Meyer, Richard Renka, David Sandvik, Nathaniel Whitney, Charles Gwinn, Edwin P. Sweet, Leonard Linde, Edward A. Johnson. A quorum was present and

the meeting was declared competent to proceed.

Dr. Saylor moved that the minutes of the previous meeting not be read inasmuch as they were published in the SOUTH DAKOTA JOURNAL OF MEDICINE. The motion was seconded and carried.

Dr. Messner introduced the following special guests: Dennis Wolf, M.D., President, North Dakota Medical Association; Thomas Briggs, M.D., President, Minnesota Medical Association; Mr. Vernon Wagner, Executive Secretary, North Dakota Medical Association.

Mrs. Marie Hovland introduced Mrs. Glenda Bates, President of the AMA Auxiliary, who spoke to the House of Delegates on the role of the Auxiliary.

Dr. Hamm, President of the South Dakota State Medical Association presented the following awards:

C. B. Alford Award — Granville Steele, M.D., Aberdeen, SD

Past President's Award — Durward Lang, M.D., Omaha, NE

Community Service Award — Paul Hohm, M.D., Huron, SD.

Distinguished Service Award — Karl Wegner, M.D., Sioux Falls, SD

50 Year Awards — Roland Hubner, M.D., Yankton, SD
C. C. Pascale, D.O., Centerville, SD

Mrs. Mollie Krafka presented the AMA-ERF check to Robert Quinn, M.D., Dean of the USD School of Medicine, on behalf of the South Dakota Auxiliary.

Dr. Messner then announced the appointments to the Nominating Committee as follows:

District # 1 — William Taylor, M.D.
District # 2 — Parry Nelson, M.D.
District # 3 — Curtis Wait, M.D.
District # 4 — R. J. Zakahi, M.D.
District # 5 — H. L. Saylor, Jr., M.D.
District # 6 — Charles D. Monson, M.D.
District # 7 — Lowell Hyland, M.D.
District # 8 — Richard Porter, M.D., Chairman
District # 9 — Robert Ferrell, M.D.
District #10 — M. George Thompson, D.O.
District #11 — Leonard Linde, M.D.
District #12 — Ed Johnson, M.D.

Appointments to the various reference committees were announced as follows:

Reference Committee on Credentials, Resolutions and Memorials, Reports of Officers and Councilors:
Ravi Kapur, M.D., Chairman
Rodney Parry, M.D.
Charles Flohr, M.D.

Reference Committee on Reports of Commissions on Medical Service, Legislation and Governmental Relations:
William G. M. Huet, Chairman
W. Nicol Guddal, M.D.
Brad Randall, M.D.

Reference Committee on Reports of Commissions on Scientific Medicine, Internal Affairs, Communications and Liaison, and Professional Liability:
Richard Holm, M.D., Chairman
T. H. Bhatti, M.D.
William Mattson, M.D.

Reference Committee on Reports of Special Committees and Miscellaneous Business:
Guy Tam, M.D.
Richard Renka, M.D.
Jeffrey Hagen, M.D.

Dr. Gere moved that the reports of the President, President-elect, Vice President, Secretary-Treasurer, Chairman of the Council, Delegate and Alternate Delegate to the AMA, Speaker of the House, Councilor at Large, Executive Secretary and Councilors not be read and referred to the appropriate reference committee. The motion was seconded and carried.

Dr. Messner called for the introduction of resolutions from the District Societies which have not been published in the Handbook.

Dr. Rodney Parry read Resolution #4, concerning CPR training in high schools. The resolution was referred to Reference Committee #2, Reference Committee on Reports of Commissions on Medical Service, Legislation and Governmental Relations.

RESOLUTION #4

TO: House of Delegates
South Dakota State Medical Association
FROM: Seventh District Medical Society
SUBJECT: A RESOLUTION endorsing the concept of mandatory training in Basic Cardiac Life Support (BCLS) for high school students and urging the state board of education to adopt a rule requiring such training.

WHEREAS, Successful resuscitation of cardiac arrest victims in the largest communities of South Dakota has become commonplace while the success rate in the small communities has remained essentially zero; and

WHEREAS recent advances in technology and EMT training have made resuscitation of cardiac arrest victims outside the hospital in rural South Dakota a real possibility;[1] and

WHEREAS cardiac arrest victims require CPR within 4 minutes;[2] and

WHEREAS, response times of rural, volunteer ambulance services range between 5 and 7 minutes; and

WHEREAS, bystander CPR has been shown to be effective; and

WHEREAS, at least 50% of the population must be trained for a system dependent on bystander CPR to be reliable;[3] and

WHEREAS, it is unrealistic to hope to achieve the "50% trained" minimum without a mandatory training program; and

WHEREAS, BCLS courses have been successfully and affordably conducted in the largest and smallest communities of the state; and

WHEREAS, the American Heart Association, Dakota Affiliate has demonstrated an ability to assure quality of instruction in BCLS training programs; and

WHEREAS, many deaths from cardiac arrest might be prevented, if bystander CPR were available;

THEREFORE, BE IT RESOLVED, by the S.D.S.M.A. that the association endorses the concept of mandatory training in Basic Cardiac Life Support for all public and nonpublic secondary school students in the state and, further, urges the state board of education to adopt rule 24:03:06:17.[4]

(FOOTNOTES)

(1) Three studies have now shown a marked increase in successful resuscitation (discharged from the hospital) of victims of cardiac arrest with an initial rhythm of ventricular fibrillation (50-60% of all cardiac arrests) when the ambulance crew was able to provide immediate defibrillation (EMT-D) compared to initial care by basic EMT's (EMT-A).

King County 1978

EMT-D	EMT-A
26% discharged	7% discharged

NEJM 1980; 302:1379-1382

* * *

Iowa 1981-1983

EMT-D	EMT-A
19% discharged	3% discharged

NEJM 1984; 310:219-223

* * *

King County 1979-1982

EMT-D	EMT-A
+ Paramedics	+ Paramedics
42% discharged	19% discharged

JAMA 1984; 251:1723-1726

(2) The Iowa study confirmed the need for prompt CPR —

figures shown here are for victims in the experimental group — all received EMT defibrillation.

	Survivors	Non-Survivors
Time to CPR	2.8 min	4.7 min

(3) Unpublished study from the University of Nebraska, Joseph Ornato, M.D., FACC, director of emergency services, U. of Nebraska.

(4) 24:03:06:17. Basic Cardiac Life Support. All public and nonpublic secondary schools shall provide and all students shall receive instruction in basic cardiac life support. The instruction shall be given according to the standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care issued by the American Heart Association August 1, 1980. The instructor shall be approved annually by the State Superintendent. The State Superintendent may certify a person as an instructor upon submission of evidence that the person is currently certified as an instructor in BCLS by the American Heart Association.

Dr. James Larson read Resolution #5, concerning the wording on the Medicare EOB form. The resolution was referred to Reference Committee #2, Reference Committee on Reports of Commissions on Medical Service, Legislation and Governmental Relations.

RESOLUTION #5

TO: House of Delegates
South Dakota State Medical Association
FROM: Watertown District Medical Society
SUBJECT: Medicare Explanation of Benefits Language

WHEREAS, Medicare has chosen to use language in its explanation of benefits that the SDSMA feels is offensive to the profession.

THEREFORE, BE IT RESOLVED that the House of Delegates voice its displeasure to the fiscal intermediary — Regional Office — and the HCFA and request this terminology be modified so as to be less degrading to the medical profession.

Dr. Jay Hubner read Resolutions #6 and #7 concerning the South Dakota Human Services Center. The two resolutions were referred to Reference Committee #4, Reference Committee on Special Committees and Miscellaneous Business.

RESOLUTION #6

TO: House of Delegates
South Dakota State Medical Association
FROM: Yankton District Medical Society
SUBJECT: Support of South Dakota Human Services Center

WHEREAS, the South Dakota Human Services Center, with an average inpatient census of 405 patients, is the only state operated short-and-long-term hospital for the mentally ill in South Dakota; and

WHEREAS, the South Dakota Human Services Center provides essential medical-psychiatric care to approximately 1,000 new admissions per year; and

WHEREAS, the South Dakota Human Services Center admits approximately 460 acutely ill psychiatric patients per year, whose average length of stay is 69 days of acute hospitalization; and

WHEREAS, there are approximately 250 long-term psychiatric patients at the South Dakota Human Services Center, who continue to need long-term inpatient psychiatric care, and

WHEREAS, the South Dakota Human Services Center operates a 20 bed unit that provides the only short-and-long-term psychiatric inpatient treatment and evaluation program for adolescents in South Dakota, and

WHEREAS, the South Dakota Human Services Center provides essential inpatient treatment programs with 57 inpatient beds for persons subject to drug and alcohol problems, and

WHEREAS, the South Dakota Human Services Center in the past six years has dramatically improved its treatment programs and quality of staff to manage these improved programs, and

WHEREAS, in the past six years significant expenditure of state monies has been provided to renovate major portions of the hospital's physical plant to meet minimal state and federal fire-safety and medical-treatment standards, and

WHEREAS, during the past six years the South Dakota Human Services Center has been developed as a Dean's Committee teaching hospital of the University of South Dakota School of Medicine, and

WHEREAS, this hospital provides essential educational opportunities to medical and nursing students of the University of South Dakota and other area colleges, and

WHEREAS, bureaucratic and legislative actions annually subject the South Dakota Human Services Center to real and threatened reductions in essential funding of state monies needed to continue the provision of these essential treatment and educational endeavors, and

WHEREAS, if the funding for the South Dakota Human Services Center is reduced significantly, severe reductions in patient care and service will occur with resulting mischief to helpless patients, and

WHEREAS, if state funds are not found to provide the essential services of the South Dakota Human Services Center, the treatment and educational programs of this hospital will severely deteriorate from the acceptable current levels;

IT IS THEREBY RESOLVED, that the South Dakota State Medical Association, by this resolution duly voted and passed during its 1984 annual meeting, expresses the Medical Association's concern for the fiscal and service well being of the South Dakota Human Services Center. By developing and passing this resolution in support of the South Dakota Human Services Center, the South Dakota State Medical Association requests that the South Dakota Board of Charities and Corrections, Governor William Janklow and the South Dakota State Legislature perform whatever actions are necessary and available to their offices to continue the high level of the current operation of the South Dakota Human Services Center now and in its future years.

RESOLUTION #7

TO: House of Delegates
South Dakota State Medical Association
FROM: Yankton District Medical Society
SUBJECT: Physician Representation on Planning Committee for South Dakota Human Services Center

WHEREAS, the South Dakota Human Services Center provides medical care and psychiatric care to mentally ill citizens of South Dakota; and

WHEREAS, staff at the South Dakota Human Services Center also provides care to men and women at the Trusty Facility and the Women's Correctional Facility; and

WHEREAS, there is consideration for an additional correctional program to be located on the grounds of the South Dakota Human Services Center; and

WHEREAS, the planning of such a program might entail the relocation, displacement or loss of certain programs and services for the mentally ill at the South Dakota Human Services Center to the detriment of those patients; and

WHEREAS, the physicians of South Dakota have a responsibility for the quality of care delivered in this state;

LET IT THEREBY BE RESOLVED, that a representative committee of physicians should participate in any planning or decision making regarding the South Dakota Human Services Center facility that would impact, in any way, on the quality or availability of patient care at that institution.

Dr. Messner then referred the reports and resolutions in the

STRETCHING THE HEALTH CARE DOLLAR

Health care coverage is meant to be used, but not abused. When some people use their coverage unwisely or unnecessarily, it costs all of us plenty in terms of higher health care costs.

It's a concern of South Dakota physicians as well as Blue Shield. By urging South Dakotans to use their health care services prudently, we can help control health care utilization.

Working together, we can stem the rising costs and get the most for our health care dollars.

SOUTH DAKOTA BLUE SHIELD
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Sioux Falls, South Dakota

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Handbook to the various reference committees.

BYLAW REVISION #1

TO: House of Delegates
South Dakota State Medical Association
FROM: Council
South Dakota State Medical Association
SUBJECT: Bylaw Change Regarding Commission Terms

ARTICLE X Commissions
Section 2. (((d. Terms — No member shall serve on a commission for more than three (3) consecutive terms of three (3) years each, and no member shall serve as chairman of a commission for more than three (3) consecutive years.)))

ARTICLE X Commissions
Section 4. d. Professional Liability Commission — The Commission on Professional Liability shall consist of nine (9) members. The commission shall be appointed by the President; the original appointments shall consist of three appointments for a one year term; three appointments for a two year term; and three appointments for a three year term. Thereafter, all appointments shall be for three year terms. (((No member shall serve on this commission for more than three (3) consecutive terms of three (3) years each, and no member shall serve as chairman of this commission for more than three (3) consecutive years.))) The commission's duties shall be to study and work in a liaison capacity and make recommendations to the Council in matters pertaining to professional liability problems and insurance coverage in the State of South Dakota.

RESOLUTION #1

TO: House of Delegates
South Dakota State Medical Association
FROM: Council
SUBJECT: Auxiliary promotion of vehicle safety restraints for children
WHEREAS, the members of the Auxiliary of the South Dakota State Medical Association have demonstrated great concern for child safety in vehicles, and
WHEREAS, the Auxiliary has successfully supported and promoted legislation requiring vehicle safety restraints for children in South Dakota,
THEREFORE BE IT RESOLVED, that the South Dakota State Medical Association commends the Auxiliary for its efforts in promoting child safety by the use of vehicle safety restraints.

RESOLUTION #2

TO: House of Delegates
South Dakota State Medical Association
FROM: Sixth District Medical Society
SUBJECT: Medicare Statement on the Face Sheet
WHEREAS, Medicare officials have insisted that a statement be placed on the face sheet of Medicare patients which threatens physicians with fines or imprisonment if diagnoses are falsified or concealed; and
WHEREAS, physicians in South Dakota feel that this statement implies that we are being threatened into keeping accurate records and is therefore highly insulting to our sense of integrity; therefore
BE IT RESOLVED, that the House of Delegates of the South Dakota State Medical Society empower its elected officers and executive staff to work with Medicare officials to have this statement removed from the charts.

Excerpt from Medicare Face Sheet

"I certify that the identification of the principal and secondary diagnoses and the procedures performed is accurate and complete to the best of knowledge. (Notice: Intentional misrepresentation, concealment, or falsification of this information may, in the case of a medical beneficiary, be punishable by imprisonment, fine or civil penalty.)"

RESOLUTION #3

TO: House of Delegates
South Dakota State Medical Association
FROM: Seventh District Medical Society
SUBJECT: Payment for collection of evidence in rape cases
WHEREAS, the cost of law enforcement is traditionally borne by all citizens through their government rather than the individual protected or victimized, and
WHEREAS, the collection of evidence after a crime is a recognized part of law enforcement, and
WHEREAS, rape represents the only major crime where the victim is expected to partially pay for the evidence collection phase of her (or his) law enforcement protection in the form of the emergency room rape examination,
THEREFORE, BE IT RESOLVED, that the Seventh District Medical Society and the South Dakota State Medical Association support and draft legislation allocating monies for at least partial reimbursement to the victims of sexual or other abuse for the evidentiary collection expenses incurred during subsequent medical examination.

Dr. William Rossing discussed the action taken by the Council of the SDSMA regarding the physician's fee freeze and asked that the recommendation be referred to a reference committee for a statement from the House of Delegates. This subject was referred to Reference Committee #4, Reference Committee on Reports of Special Committees and Miscellaneous Business.

Dr. Messner made several announcements concerning the schedule for the annual meeting.

The meeting adjourned at 9:30 a.m.

MINUTES OF THE SECOND HOUSE OF DELEGATES MEETING

9:30 A.M. Washington Room, Howard Johnsons
Sunday, June 10, 1984 Rapid City, South Dakota

The meeting was called to order by Frank Messner, M.D., Speaker of the House. Those present for roll call were Doctors Joseph Hamm, Howard Saylor, Richard Gere, W. O. Rossing, Frank Messner, Gerald Tracy, Bruce Lushbough, G. H. Steele, J. A. Eckrich, Jr., James Larson, Curtis Wait, David Buchanan, C. D. Monson, Richard Gere, K. Gene Koob, Richard Gunnarson, Lowell Hyland, Richard Porter, Jay Hubner, Thomas Krafka, Robert Ferrell, James Jackson, M. George Thompson, W. R. Taylor, Winston Odland, G. J. Bloemendaal, Parry Nelson, W. N. Guddal, Tad Jacobs, Ravi Kapur, Wm. G. M. Huet, Charles Flohr, Jerry Freeman, T. H. Bhatti, George Bruins, Rodney Parry, Guy Tam, Brad Randall, Jerry Simmons, David Hoversten, Anthony Javurek, James Quale, Jeffrey Hagen, Roger Stoltz, Morris Radack, David Smith, Larry Meyer, Richard Renka, Robert Williams, Dale Berkebile, Nathaniel Whitney, C. B. Gwinn, E. P. Sweet, Leonard Linde and E. A. Johnson.

Dr. Odland moved to dispense with the reading of the minutes of the previous meeting inasmuch as they will be published and distributed. The motion was seconded and carried.

Dr. Messner introduced Dr. Frank Jirka, AMA President. Dr. Jirka reviewed AMA activities during the past year, in particular legislation which has been introduced which would affect the practice of medicine, and the AMA's call for a one year freeze on physicians fees. He issued two challenges to the physicians: 1) to develop new ideas to help control health care costs and 2) to effectively oppose government and/or business proposals which will hurt physicians and their patients. He recognized the various types of physician practices and encouraged the State Medical Association to develop programs which appeal equally to all physicians in our state. Dr. Jirka stated he feels the greatest challenge to physicians is 1) to ensure that the quality of care is the highest, and 2) to assure that care is available and accessible.

Dr. Richard Porter read the report of the Nominating Committee.

REPORT OF THE NOMINATING COMMITTEE

The Nominating Committee submits the following recommendations for the consideration of the House of Delegates:

MED SCIENCES LIBRARY

OFFICERS	
President Elect	Richard G. Gere, M.D.
Vice President	William O. Rossing, M.D.
Speaker of the House	Frank D. Messner, M.D.
AMA Delegate	Gerald E. Tracy, M.D.
AMA Alternate Delegate	Bruce C. Lushbough, M.D.
COUNCILORS — 3 year terms	
Aberdeen District #1	J. A. Eckrich, Jr., M.D.
Brookings-Madison District #3	Curtis Wait, M.D.
Huron District #5	David Buchanan, M.D.
Mitchell District #6	C. D. Monson, M.D.
Sioux Falls District #7	Michael Pekas, M.D.
	Lowell Hyland, M.D.
Yankton District #8	Jay Hubner, M.D. (2 year term)
Black Hills District #9	Ed James, M.D.
ALTERNATE COUNCILORS — 3 year terms	
Aberdeen District #1	Juan Chavier, M.D.
Huron District #5	G. Robert Bell, M.D.
Sioux Falls District #7	Dennis Johnson, M.D.
	D. G. Ortmeier, M.D.
Yankton District #8	John Sternquist, M.D. (1 year term)
Black Hills District #9	Thomas Krafka, M.D.

ANNUAL MEETING SITE
1985 — Sioux Falls, SD, June 6, 7, 8, 9, 1985
1986 — Rapid City, SD, June 5, 6, 7, 8, 1986
1987 — Sioux Falls, SD

Respectfully submitted,
NOMINATING COMMITTEE
Richard Porter, M.D., Chairman
William R. Taylor, M.D.
Parry Nelson, M.D.
Curtis Wait, M.D.
R. J. Zakahi, M.D.
H. L. Saylor, M.D.
C. D. Monson, M.D.
Lowell Hyland, M.D.
Robert Ferrell, M.D.
George Thompson, D.O.
Leonard Linde, M.D.
Ed Johnson, M.D.

Dr. Messner called for nominations from the floor. There being no nominations from the floor, **Dr. Hamm moved to elect the officers, councilors and alternate councilors as submitted and to accept the report of the Nominating Committee. The motion was seconded and carried.**

Dr. Ravi Kapur read the report of the Reference Committee on Credentials, Resolutions and Memorials and Reports of Officers and Councilors.

REPORT OF THE REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND MEMORIALS AND REPORTS OF OFFICERS AND COUNCILORS

The following delegates, alternate delegates, officers and councilors of the South Dakota State Medical Association were present: Drs. Joseph N. Hamm, Howard L. Saylor, Jr., Richard Gere, William O. Rossing, Frank Messner, Gerald E. Tracy, Bruce Lushbough, Durward Lang, Jerome A. Eckrich, Jr., James Larson, Curtis Wait, R. Curtis Jahraus, David Buchanan, Charles D. Monson, Michael Pekas, Denny Ortmeier, Richard E. Gunnarson, Lowell Hyland, Richard Porter, Jay Hubner, Thomas L. Krafka, Robert L. Ferrell, James Jackson, M. George Thompson, Joseph Kass, William R. Taylor, Winston Odland, Parry Nelson, W. Nicol Guddal, Richard Holm, Tad Jacobs, Ravi Kapur, William G. M. Huet, Charles Flohr, Gary Van Ert, James Ryan, Anthony J. Javurek, George Bruins, Dorence L. Ensberg, Jerry Simmons, James Quale, David Hoversten, Bradley Randall, Rodney Parry, Jeffrey B. Hagen, Jerome W. Freeman, George Barnett, Guy Tam, Morris Radack, Larry Meyer, Richard Renka, David Sandvik, Edwin P. Sweet, Leonard Linde, Edward A. Johnson, Theodore Sattler, Nathaniel Whitney and Charles Gwinn.

A quorum was present for the meeting of the House of Delegates. Total registration for the convention is 325, including 204 physicians, 11 guests, 83 Auxiliary members and 27 sponsors.

The committee submits the following resolution for the consideration of the House of Delegates:

WHEREAS, the Black Hills District Medical Society, the Black Hills District Auxiliary, the Aberdeen District Auxiliary and the Mitchell District Auxiliary members have been so thorough in making arrangements for the success of the combined meeting of our Annual Meeting,

BE IT RESOLVED, that the South Dakota State Medical Association give its voice in appreciation and thanks to the local physicians in the Black Hills District and the members of the Black Hills District, the Aberdeen District and the Mitchell District Auxiliaries.

WHEREAS, the management of the Howard Johnson Motor Lodge has been so cooperative in providing facilities for the success of the Annual Meeting of the South Dakota State Medical Association,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks and appreciation to the Howard Johnson Motor Lodge.

WHEREAS, the *Rapid City Journal,* KOTA TV and radio, KIMM radio, KKLS radio AM and FM, KSQY radio, the United Press International and the Associated Press have been most cooperative in presenting the public news of the annual meeting of the South Dakota State Medical Association,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks to the *Rapid City Journal,* KOTA TV and radio, KIMM radio, KKLS radio AM and FM, KSQY radio, the United Press International and the Associated Press.

WHEREAS, the Arrowhead Country Club has been most cooperative in providing facilities for the golf tournament and Thursday evening stag party and the Auxiliary luncheon on Saturday,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks and appreciation to the Arrowhead Country Club.

BE IT RESOLVED, that $50 be donated to the South Dakota Medical School Endowment Association in memory of the following physicians who died during the past year:

R. W. Honke, M.D.
Vernon Cutshall, M.D.
Howard Wold, M.D.
A. J. Barrett, M.D.
C. J. McDonald, M.D.
E. A. Rudolph, M.D.
Robert Donahoe, M.D.
C. F. J. Blunck, M.D.

The committee reviewed the reports of the officers and councilors and recommends they be accepted as submitted.

The committee would like to specifically recognize the University of South Dakota School of Medicine, its work in supporting community and state awareness of medicine, continuing medical education, and in elevating quality of medical care. The committee recognizes these accomplishments were achieved despite imposed budget restraints.

The committee would also like to recognize the superb and outstanding work and ability of our Executive Secretary, Robert Johnson and his staff during the last year in conducting the business of the South Dakota State Medical Association.

Respectfully submitted,
REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND REPORTS OF OFFICERS AND COUNCILORS
Ravi Kapur, M.D., Chairman
Rodney Parry, M.D.
Charles Flohr, M.D.

Dr. Flohr moved to correct the report to include Dr.

Charles Flohr and Dr. Gary Van Ert in the roll call. The motion was seconded and carried. Dr. Flohr moved to accept the report of the Reference Committee on Credentials, Resolutions and Reports of Officers and Councilors as amended. The motion was seconded and carried.

Dr. William G. M. Huet read the report of the Reference Committee on Reports of the Commission on Medical Service and the Commission on Legislation and Governmental Relations.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations. The Reference Committee recommends acceptance of this report.

The Reference Committee reviewed the report of the Commission on Medical Service. The Reference Committee recommends the acceptance of the report of the Commission on Medical Service.

The following resolutions were presented to the Reference Committee:

Resolution #1 — The Reference Committee recommends the House of Delegates adopt this resolution with the following addition: The County Health Nurse Offices in the state provide safety restraints for children in cars for needy individuals. John N. Jones, Division of Health Services in Pierre, may be contacted for information on this service.

Dr. Guddal moved that the House accept the recommendation of the reference committee to adopt Resolution #1 with the addition. The motion was seconded and carried.

Resolution #4 — The Reference Committee recommends the House of Delegates adopt this resolution with the following addition: The State Health Department has office facilities and services available for CPR training to the SDSMA should they be needed. Contact John N. Jones, Division of Health Services in Pierre, for information. The AHA, Dakota Affiliate, has over 700 certified instructors willing to help teach this program on a voluntary basis.

Dr. Gwinn moved that the House accept the recommendation of the reference committee to adopt Resolution #4 with the addition. The motion was seconded and carried.

Resolution #5 was presented by the Watertown District Medical Society. The Reference Committee recommends adoption of this resolution.

Dr. Larson moved that the House adopt Resolution #5 introduced by the Watertown District Medical Society. The motion was seconded and carried.

Respectfully submitted,
REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS
William G. M. Huet, M.D., Chairman
W. Nicol Guddal, M.D.
Brad Randall, M.D.

Dr. Hamm moved to accept the report of the Reference Committee on Reports of the Commission on Medical Service, and the Commission on Legislation and Governmental Relations. The motion was seconded and carried.

Dr. C. B. Gwinn read the report of the Reference Committee on Reports of the Commissions on Scientific Medicine; Internal Affairs, Communications and Liaison; and Professional Liability.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSIONS ON SCIENTIFIC MEDICINE, INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON, AND PROFESSIONAL LIABILITY

The Reference Committee reviewed the report of the Commission on Scientific Medicine. The Reference Committee recommends acceptance of this report.

The Reference Committee reviewed the report of the Commission on Internal Affairs, Communications and Liaison. The Reference Committee recommends acceptance of this report.

The Reference Committee reviewed the report of the Commission on Professional Liability. The Reference Committee recommends acceptance of this report.

The Reference Committee wishes to commend the members of these three Commissions for their work on behalf of the members of the State Medical Association during the past year.

The Reference Committee reviewed the proposed amendments to the Bylaws of the South Dakota State Medical Association. The Reference Committee recommends adoption of these Bylaw changes which limit the number of terms a commission member may serve on a commission and the number of years a commission member may serve as chairman of that commission.

Respectfully submitted,
Richard Holm, M.D., Chairman
Charles Gwinn, M.D.
T. H. Bhatti, M.D.

Dr. Smith moved to accept the report of the Reference Committee on Reports of the Commissions on Scientific Medicine; Internal Affairs, Communications and Liaison; and Professional Liability. The motion was seconded and carried.

Dr. Guy Tam read the report of the Reference Committee on Reports of Special Committees and Miscellaneous.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS

The Reference Committee has reviewed and recommends acceptance of reports from the Committee for Continuing Medical Education, the Grievance Commission, the Long Range Planning Committee, the Liaison Committee, the South Dakota Political Action Committee, the Board of Directors of the South Dakota Medical School Endowment Association, the

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president of the South Dakota Medical School Endowment Association and the Archives and History Commission. In this important election year of 1984 the Reference Committee recommends that the House of Delegates encourages active participation in the political process by physicians and spouses and membership in SoDaPAC.

The Reference Committee recommends that the House of Delegates endorse the Council action of April 1984 calling for a voluntary one year freeze on physicians' fees, and supports the challenge to other independent practitioners, health care providers and suppliers to do likewise.

The Reference Committee recommends adoption of Resolution #2 concerning the Medicare statement on the face sheet.

Dr. Gere moved to accept the reports of the special committees, to endorse the Council's action calling for a voluntary one year freeze on physicians' fees and to adopt Resolution #2. The motion was seconded and carried.

The Reference Committee recommends adoption of Resolution #6 which was introduced by the Yankton District Medical Society.

Dr. Buchanan moved that Resolution #6 submitted by the Yankton District Medical Society requesting support for the South Dakota Human Services Center be referred to the Council for revision and further consideration. The motion was seconded and carried.

The Reference Committee recommends adoption of Resolution #7 submitted by the Yankton District Medical Society.

Dr. Williams moved to accept Resolution #7 introduced by the Yankton District Medical Society calling for physician representation on any committee involved in planning for or making decisions about the South Dakota Human Services Center. The motion was seconded and carried.

The Reference Committee by a majority vote recommend adoption of Resolution #3 concerning payment for collection of evidence in rape cases.

Dr. Freeman moved that the House adopt Resolution #3. The motion was seconded and carried.

Respectfully submitted,
REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS
Guy Tam, M.D., Chairman
Jeffrey Hagen, M.D.
Richard Renka, M.D.

Dr. Ferrell moved to accept the report of the Reference Committee on Reports of Special Committees and Miscellaneous Business as amended. The motion was seconded and carried.

Dr. Gerald Tracy moved that the House of Delegates recognize Dr. M. C. Thompson of Watertown in his 50th year since graduation from the University of South Dakota School of Medicine. The motion was seconded and carried.

Dr. Tracy also reported to the House of Delegates that he visited with Dr. Russell Harris, a past president of the State Medical Association, who is currently recuperating at home and extended greetings to the House from Dr. Harris.

Dr. Messner administered the oath of office to Dr. Howard Saylor, the 1984-85 President. Dr. Hamm thanked the House members for their assistance during his presidential year and he then presented the presidential medallion to Dr. Saylor. Dr. Saylor addressed the House of Delegates briefly and asked for the assistance of the members during the coming year.

Dr. Messner introduced the new officers and councilors and recognized Dr. Hamm for his outstanding service to the Association during the past year.

The meeting adjourned at 10:40 a.m.

REPORT OF THE PRESIDENT AND CHAIRMAN OF THE EXECUTIVE COMMISSION

With AMA Delegate, Gerald E. Tracy, M.D., AMA Alternate Delegate, Bruce C. Lushbough, M.D., and Executive Secretary, Bob Johnson, I attended the Interim and the Annual Meetings of the AMA. During both of these sessions, we represented you to the North Central Conference which includes delegations from Iowa, Minnesota, Nebraska, North Dakota and Wisconsin. The Conference affords an opportunity to emphasize our concerns to the AMA.

With the Executive Secretary, Bob Johnson, and with Lorin Pankratz since he joined the staff, I enjoyed attending meetings with each of the district societies. These visits are one of the most gratifying assignments of the president. Some districts are very well organized and active. A few might consider restructuring their activity.

With the Executive Secretary, I met with Attorney General Meierhenry and Secretary of Health Massa to review the position of the Association regarding matters of medical policy at the state level.

The Executive Commission retreat was held at Peters' Sunset Beach, Glenwood, Minnesota, in August, 1983. The Commission met in Sioux Falls on January 4, 1984, and held a joint meeting with the Board of Directors of the South Dakota Hospital Association in November of 1983. All matters discussed were submitted to the Council for consideration.

An Ad Hoc Committee was appointed by the president at the direction of the Council to meet with representatives of the South Dakota State Bar Association. The discussion revolved around the relationships of lawyers and physicians which would benefit clients or patients. Both delegations will report to their respective Associations for further direction.

Your president is especially grateful to the affable, accessible and accommodating executive staff. Their dedication and skill deserve the highest praise. The many activities of your Association would falter without them and I commend them to the membership.

Respectfully submitted,
Joseph N. Hamm, M.D.
President and Chairman
Executive Commission

The Reference Committee reviewed the report of the President and Chairman of the Executive Commission and recommends it be accepted as submitted.

REPORT OF THE PRESIDENT-ELECT

As President-Elect of the Medical Association, I have attended all of the meetings of the House of Delegates, Council and the Executive Commission during the past year. It has been a constant source of pride and satisfaction to see the commitment and the energy that the various members of the commissions, council and bodies of the State Medical Association put into their deliberations and conclusions.

In February of 1984, I attended the AMA Leadership Conference in Chicago, during which time, in addition to the regular programs that were presented, I did have an opportunity to meet and discuss with members of our surrounding state medical societies many of the problems that we are concerned with. We are all having the same problems, and hopefully, through continued cooperation of the membership and help from the AMA, we can help solve some of these.

All of the functions of our Medical Association are very well coordinated by the staff members without whom we would not be able to continue on a high plane. I am looking forward to serving the Association as President in the coming year and want you to know that I always have and always will consider this your association, with an open door and an open ear for any suggestions or help that you may give.

Respectfully submitted,
Howard L. Saylor, Jr., M.D.
President-Elect

The Reference Committee reviewed the report of the President-Elect and recommends it be accepted as submitted.

REPORT OF THE VICE PRESIDENT

As Vice President of the South Dakota State Medical Association, I have attended all the meetings of the Council and Executive Commissions of the Medical Association and Ad Hoc Committee on Medicaid. With the ever changing practice of medicine, the value of these meetings becomes more apparent. It is gratifying to see that year after year the attendance remains

high, and the quality of all the members of the Council and Commissions remain excellent.

Respectfully submitted,
Richard G. Gere, M.D.
Vice President

The Reference Committee reviewed the report of the Vice President and recommends it be accepted as submitted.

REPORT OF THE SECRETARY-TREASURER

The 1983-84 fiscal year of the SDSMA is concluding with the Association again showing a strong financial position. Investment income from the various financial market instruments, captured in earlier years when interest rates were considerably higher than currently obtainable, continues to be an important component of this equation. Consequently, no dues increase to the membership has had to be considered during this period. Growth in membership throughout the state also adds a significant dollar amount to the operational margin enjoyed this year. Again, it would be appropriate to point out benefits we continue to enjoy from the well-balanced and prudent decisions provided by our Executive Secretary in this area.

During the year I have attended meetings of the Council and the Executive Committee on a regular basis, participated in joint planning sessions with the S.D. Hospital Association, and attended the AMA North Central Regional Conference last fall. Additionally, I have represented the Association in several brief public affairs media events generated in the Sioux Falls area.

The Secretary-Treasurer also serves as an appointed member of the Health Professions Loan Advisory Board of the State of South Dakota. No meetings were convened by the body, however, during 1983.

Respectfully submitted,
W. O. Rossing, M.D.
Secretary-Treasurer

The Reference Committee reviewed the report of the Secretary-Treasurer and recommends it be accepted as submitted.

REPORT OF THE CHAIRMAN OF THE COUNCIL

The Council of the South Dakota State Medical Association met at routinely scheduled intervals. The topics continue to increase with the degree to which outside influences affect the practicing physicians in the state.

The Commissions and their respective chairmen are again to be commended for streamlining matters to come before the Council.

We continue to be represented in superior fashion by our Executive Secretary, Robert Johnson, and his very capable staff. I wish to thank them very much, both personally and through the Council.

Respectfully submitted,
Robert L. Ferrell, M.D.
Chairman, Council

The Reference Committee reviewed the report of the Chairman of the Council and recommended it be accepted as submitted.

REPORT OF THE AMA DELEGATE

It has been my pleasure to represent the South Dakota State Medical Association at both the Interim and the annual American Medical Association meetings in July and December of 1983. The meetings, as always, seemed pertinent to the regular affairs of physicians in the state of South Dakota. The Executive Secretary, Mr. Robert Johnson, has been eminently helpful in making all arrangements for these meetings. Also have had occasion to visit Washington, D.C. and to discuss with our legislators, particularly Congressman Tom Daschle, the issue of mandatory assignment. This in early April then came on the floor of the House and Mr. Daschle not only voted against it as we requested but also spoke on the floor of the House against this amendment.

These and many other issues come before the AMA on a regular basis, and I am confident they are of great support and great help in protecting the private free enterprise practice of medicine.

While there are many changes going on in the scenario of American medicine today, I think the AMA stands ready and able, as always, to support to whatever degree as humanly possible this continued need for free private enterprise practice of medicine. Again, it has been my pleasure to serve as delegate and I look forward to attending the 1984 sessions as a delegate from South Dakota.

I also attended a North Central Medical Conference primarily concerned with the economics of medicine today. These reports have all been sent to the membership and I would be happy to discuss with any members of our South Dakota State Medical Association any of the issues that come before the AMA at any time.

Respectfully submitted,
G. E. Tracy, M.D.
Delegate to the AMA

The Reference Committee reviewed the report of the AMA Delegate and recommended it be accepted as submitted.

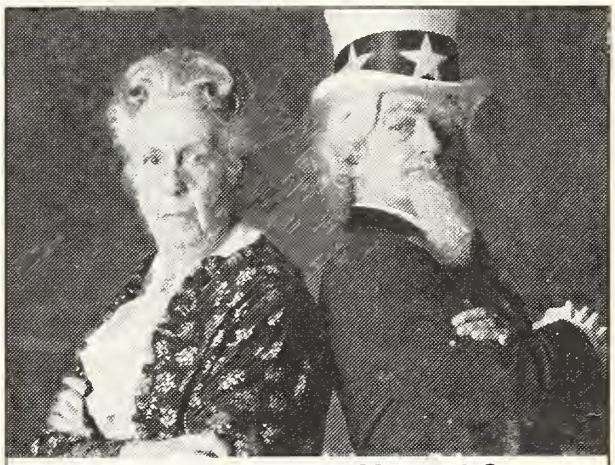
REPORT OF THE AMA ALTERNATE DELEGATE

As the AMA Alternate Delegate, I have attended meetings of the Council and Executive Commissions during the past year. I also attended the Annual Interim meetings of the AMA in 1983 and the North Central Medical Conference in November, 1983.

As new challenges confront all of us to protect our freedom in practicing medicine in ways to best serve our patients, I am convinced that we all must remain active voices in our district, state and medical societies and also in the AMA.

Respectfully submitted,
Bruce C. Lushbough, M.D.
AMA Alternate Delegate

The Reference Committee reviewed the report of the AMA Alternate Delegate and recommended it be accepted as submitted.



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REPORT OF THE SPEAKER OF THE HOUSE

I am looking forward to presiding at the upcoming meeting of the House of Delegates at the State Medical Convention in Rapid City.

I appreciate the help and support of the people responsible for the preparation of this meeting. I particularly wish to thank all the physicians who have agreed to participate in the reference committees and especially the chairmen of those committees.

The active participation through the reference committee system is important to the issues and both enhances and speeds the work of the House of Delegates.

As Speaker of the House I have attended the scheduled Council meetings and participated with the Executive Commission.

Respectfully submitted,
Frank D. Messner, M.D.
Speaker of the House

The Reference Committee reviewed the report of the Speaker of the House and recommended it be accepted as submitted.

REPORT OF THE COUNCILOR AT LARGE

It has been my privilege to serve as Councilor at Large for the past year. I have attended most of the Council meetings and Executive Committee meetings during that time.

It has been my pleasure to serve on your behalf as Councilor at Large during the past year.

Respectfully submitted,
Durward M. Lang, M.D.
Councilor at Large

The Reference Committee reviewed the report of the Councilor at Large and recommended it be accepted as submitted.

REPORT OF THE EXECUTIVE SECRETARY

It has been my distinct privilege to accompany Dr. Hamm on his visits to all twelve district medical societies this past year. Dr. Hamm has stated on several occasions that these visits were one of the highlights of his presidency and I most heartily concur. Your generosity and hospitality are unexceeded.

Again 1983 and 1984 found very heavy workloads for many of our commissions and committees, and their chairmen and members deserve a special word of thanks for their efforts on behalf of the medical profession and the people of South Dakota.

During this past year the Commission on Long Range Planning conducted a survey of the membership. Fifty-three percent of our members responded to this survey. After reviewing the responses, the commission is now embarking on additional studies in certain areas to determine appropriate changes in Association activities. It is the hope of the commission that through this survey and other such studies that they will be able to keep the Association's activities responsibe to the desires and needs of the membership.

The 1984 legislative session was, for the most part, relatively peaceful and did not adversely affect the medical profession. Of the over 20 bills that affected physicians in one way or another, all of those that the Association opposed were defeated and many of the bills we supported were adopted by our legislature. There were several bills that we followed which merit some individual discussion. On the final day of this legislative session, the legislature passed House Concurrent Resolution 1046 commending the AMA on their request to physician members to voluntarily freeze their fees, and they encouraged South Dakota physicians to do the same. Because of the concern among our legislators about health care costs, I believe that cost containment issues likely will receive considerable attention in the 1985 legislative session. The bill to license radiologic technologists was defeated, but it prompted the Chairman of the Senate Health and Welfare Committee, based upon committee discussion, to write to the Secretary of Health asking for further verification that x-ray procedures are being performed safely in South Dakota. One of our Commissions will be studying this further and formulating recommendations.

On the national level this past year we have seen two attempts to mandate assignments under the Medicare program for all hospital in-patients. A second attempt to mandate assignments was overwhelmingly rejected by the House of Representatives recently. Had such irresponsible legislation passed, it would have placed hospitals in an enforcer role and would have inappropriately established participation in a government financed health program as an essential in granting staff privileges to physicians, rather than utilizing appropriate standards such as training, experience and competence.

Most of the observers of the Washington scene feel that Congress will spend significant time in 1985 attempting to overhaul the Medicare program. In this regard, the leadership of physicians is essential if responsible solutions to the increasing cost of health care are to be found.

During this past year we have seen the implementation of the DRG's and all of the problems and frustrations they entail. The law enacting DRG's contains many onerous provisions, one of which requires the Secretary of Health and Human Services to report to Congress as to the advisability and feasibility of including physician reimbursement under the DRG's.

Health care costs are often debated in a very emotional and sometimes irresponsible fashion by politicians, the media, and the public. With this being an election year, it is more important than ever that every physician and every spouse become actively involved in the election process. Our voices must be heard now, before it's too late. I can think of no better way for a physician and his spouse to become involved than to attend your district medical society meetings regularly, become involved in your Association activities at all levels, get to know and work in campaigns of candidates for elective office (both state and national), join and support SoDaPAC, and of course, vote. Be assured our opponents will do no less.

Your Association is numerically and financially strong and blessed with outstanding leadership from your officers, councilors, and commission members. What is needed now is the commitment and involvement from the entire membership.

The officers and councilors of your association should be commended for their insight, dedication, and leadership, for they have truly represented medicine in an exemplary fashion.

We were all saddened by the loss of A. J. Barrett, M.D., our Speaker of the House. His wisdom and wit will be truly missed.

Words don't seem adequate to express the gratitude I feel for the job done by Dr. Joe Hamm, your president. He has been an outstanding ambassador for the medical profession and I count both he and his lovely wife, Dorothy, among my dearest of friends.

Respectfully submitted,
Robert D. Johnson
Executive Secretary

The Reference Committee reviewed the report of the Executive Secretary. The Committee recognized the superb and outstanding work and ability of our Executive Secretary, Robert Johnson, and his staff during the last year in conducting the business of the South Dakota State Medical Association.

REPORT OF THE FIRST DISTRICT COUNCILOR

The First District Medical Society continued to hold some monthly meetings during the winter months. The October meeting featured a talk sponsored by Lederly Laboratories and given by Elliot Frank, M.D. on the use of new Beta-Lactam antibiotics.

New officers were elected during the December 7 meeting and they are as follows:

President: Dr. Hart
Vice-President: Dr. Brown
Secretary-Treasurer: Dr. Redmond

The meeting held on January 4, 1984, featured a talk by Barry Welge, M.D. on "Calcium Channel Blocking Agents."

During the meeting on February 1, 1984, Dr. Jay Bachmayer was elected to fill the seat on the Board of Censors vacated by Dr. Christopher. Also, a nominating committee was formed for the purpose of putting together a list of delegates, alternate delegates, nominee for nominating committee, and nominee for councilor and alternate councilor. The vote will be held at the next district meeting. Upon conclusion of the business meeting, a presentation was given by the Edward D. Jones Corp. concern-

ing tax advantage investments.

A winter storm caused the cancellation of the March meeting.

The April meeting will feature a presentation by Robin Nelson, D.V.M.

A presentation sponsored by the A. H. Robbins Company regarding "Esophagitis" will be featured at the May meeting.

Respectfully submitted,
J. A. Eckrich, Jr., M.D.
G. H. Steele, M.D.
Councilors, First District

The Reference Committee reviewed the report of the Councilors from the First District Medical Society and recommended it be accepted as submitted.

REPORT OF THE SECOND DISTRICT COUNCILOR

The second district met at monthly intervals from September through May as described in their bylaws. We were honored on our October 4 meeting by the presence of the President of the South Dakota State Medical Association, Dr. Joe Hamm, and the Executive Secretary, Robert Johnson, for their annual visitation. Other monthly meetings included discussions of socio-economic factors affecting medicine and medical topics of interest to the group. Attendance was excellent during the year with reports being generated by the members of the district who participate in other activities of the State Medical Association as well as allied groups.

In addition, officers elected at the December meeting included: Dr. Paul Frazer of Clear Lake as President; Dr. Clark Likness of Watertown as Vice-President; and Dr. Gerald Tracy of Watertown as Secretary.

Respectfully submitted,
James C. Larson, M.D.
Councilor, Second District

The Reference Committee reviewed the report of the Councilors from the Second District Medical Society and recommended it be accepted as submitted.

REPORT OF THE THIRD DISTRICT COUNCILOR

Since my last report, the Third District has continued to have regular meetings with the district holding six meetings.

March 14, 1983 — Starlite Inn — Brookings. The Society met and conducted its regular business meeting plus having a topic presented by Dr. Richard P. Holm about the use and abuses of tricyclic antidepressants.

June 23, 1983 — Grandview Supper Club — Madison. This meeting entailed a rather extensive discussion of the cost containment issue and the paper on Obstetrical and Newborn Care that had been directed toward the districts by the South Dakota Medical Council. Following recommendations, Dr. Lampert was instructed to carry the feelings of the District to the Council at their next regular meeting.

March 18, 1983 — Heritage House — Arlington. Business meeting plus topic "Acute Effects of Alcohol on Balance Mechanisms" by Dr. Robert Rietz of Brookings.

October 13, 1983 — Starlite Inn — Brookings. Dr. Chutka, geriatrician from the Mayo Clinic, presented the topic "Sleeping Disorders of the Elderly." At the business meeting there was a rather long discussion about health care for the unemployed and needy that had been prompted by the letter from the American Medical Association which had been distributed to the districts.

December 1, 1983 — Annual Meeting — Flandreau Indian School — Flandreau. This also was the annual presidential visit, and Dr. Joe Hamm and Mr. Bob Johnson of the South Dakota Medical Association were present and gave an excellent discussion of the status of medicine in the state.

The following officers were elected for the next term: President — Dr. Rich G. Sample, Madison; Vice President — Tad Jacobs, D.O., Flandreau; and Secretary-Treasurer — Richard Holm, M.D., Brookings.

February 15, 1984 — Grandview Supper Club — Madison. This is a regular business meeting. Mr. Ben Johnson and Mr. Peter Galindo from South Dakota Blue Shield were present and spoke about Blue Shield and related topics. At the business meeting Dr. Curtis Wait was named Councilor. Delegates to the annual meeting were elected as Tad Jacobs and Rick Holm. The district also voted to submit Dr. B. T. Otey's name and Dr. Walt Patt's name to the Council for election as honorary life members to the South Dakota Medical Association.

Respectfully submitted,
Arthur A. Lampert, M.D.
Councilor, Third District

The Reference Committee reviewed the report of the Councilor from the Third District Medical Society and recommended it be accepted as submitted.

REPORT OF THE FOURTH DISTRICT COUNCILOR

The Fourth District Medical Society met on January 17, 1984, for its annual meeting. At this time, Dr. Joe Hamm made his annual presidential visit. Election of officers was held, and the new slate is: Dr. R. J. Owens, President, Dr. M. R. Cosand, Secretary, Dr. R. J. Zakahi, Delegate, and Dr. D. W. Borth, Alternate Delegate. The remainder of the meeting was taken up by remarks from both Dr. Hamm and Bob Johnson pertinent to the upcoming session.

The other district activities include nearly 100% participation of the local district members in the annual high school physicials, both in the spring and again in the fall.

In cooperation with the In-Service Education Department of St. Mary's Hospital, the following CME programs were sponsored:

03-15-83 — Cataract and Cataract Surgery by Dr. Henry Reichert
04-19-83 — Cardiac Rehabilitation by Dr. A. J. Tieszen
05-17-83 — Cancer Pain Management by Dr. Robert Marschke
08-16-83 — Low Back Pain by Dr. B. Krueger
09-20-83 — Headache by Dr. Harlan Payne
10-25-83 — Abdominal Trauma by Dr. M. R. Cosand
11-15-83 — Laparoscopy Experiences at St. Mary's Hospital by Dr. B. O. Lindbloom
01-17-84 — Arthritis Update by Dr. Scott Kayle
02-21-84 — Care of the Burn Patient by Dr. Lynn Solem
03-20-84 — AIDS and Related Infections by Dr. David Ohrt

Respectfully submitted,
R. C. Jahraus, M.D.
Councilor, Fourth District

The Reference Committee reviewed the report of the Councilor from the Fourth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE FIFTH DISTRICT COUNCILOR

The Huron District Medical Society met regularly during the past year with both business and scientific meetings.

There was no loss of membership, but two new members were added; namely, Dr. Fred Schneider and Dr. Bernie Heilman. There are now 30 members in our district.

The new officers for 1984 are Dr. Ravi Kapur, President, Dr. Tom Dean, Vice President, Dr. Theodore A. Hohm, Secretary-Treasurer. The delegates are Dr. Ravi Kapur and Dr. Tom Dean, and the alternate delegates are Dr. William Huet and Dr. Theodore A. Holm. Dr. David Buchanan was selected as Councilor.

Respectfully submitted,
David Buchanan, M.D.
Councilor, Fifth District

The Reference Committee reviewed the report of the Councilor from the Fifth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE SIXTH DISTRICT COUNCILOR

The Sixth District of the South Dakota State Medical Association met on a fairly regular basis this year. Each meeting had various members present and interesting and/or unusual case-history from their own practice, in addition to the usual social

and relaxation aspect.

Officers for the Sixth District were as follows: President, Timothy J. Judge, M.D.; Vice-President, John B. Jones, M.D.; Secretary-Treasurer, Sandro Visani, M.D.; Delegates, Charles E. Flohr, M.D.; Anthony J. Silvagni, D.O., and Judson O. Mabee, M.D.; and Alternates, Maynard H. Porter, M.D., and Walter Baas, M.D. Our district has now reached a total membership of 49.

Throughout the year, two of our main objectives were: 1) to improve the rapport and cooperation among all the district's members; and 2) keeping abreast of political issues in medicine today.

Looking ahead to the continued success of the South Dakota Medical Association, we of the Sixth District hope to stimulate interest in our district, as well as improvement in the quality, regularity, and attendance of our meetings.

Respectfully submitted,
Charles D. Monson, M.D.
Councilor, Sixth District

The Reference Committee reviewed the report from the Councilor for the Sixth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE SEVENTH DISTRICT COUNCILORS

The 1984 District Seven President is under the very able leadership of Rodney Parry, M.D. (Pulmonary Medicine). Jerome Freeman, M.D. (Neurology) serves as the current District Seven Secretary.

The 1983 calendar year District Seven President was Gail Benson, M.D. (Orthopedics). The 1983 District Seven Secretary was Jeffrey Hagen, M.D. (Pathology).

According to Mrs. Jan Anderson, the Assistant Executive Secretary of the South Dakota State Medical Association, the 1982 District Seven membership census was 208. The 1983 District Seven membership census is 222. Current membership census statistics for calendar year 1984 are presently being compiled and incomplete at the time of this writing. Appropriate officers' names for the Executive Committee, the Council, and the House of Delegates including alternates are listed in other sections of this publication.

Communities included in District Seven are Bridgewater, Canton, Colton, Dell Rapids, Freeman, Salem, Sioux Falls, Valley Springs and Viborg. Current hospital administrators in District Seven are as follows: Canton, John Devick; Dell Rapids, Donald Olson; Freeman, Larry Ravenberg; Sioux Falls, Crippled Children's Hospital and School, E. B. Morrison, Ph.D., McKennan Hospital, Roger Paavola, Sioux Valley Hospital, Lyle Schroeder, Veteran's Administration Center, Dexter D. Dix; Viborg, Warren Kuhler.

District Seven Hospital Chiefs of Staff for 1984 are as follows: Canton, Eugene Regier, M.D. (Family Practice); Dell Rapids, K. Gregg Peterson, M.D. (Pathology); Freeman, Dennis Epp, M.D. (Family Practice); Sioux Falls, Crippled Children's Hospital and School, Robert E. VanDemark, Sr., M.D. (Orthopedics), McKennan Hospital, Neil Elkjer, M.D. (Family Practice), Sioux Valley Hospital, K. Gene Koob, M.D. (Neurology), Veterans Administration Center, George W. Wyatt, M.D. (Ob-Gyn); Viborg, Earl Nelson, M.D. (Family Practice).

Meeting times for District Seven are in the months of September through May and fall on the first Tuesday of the month. Activities relevant for District Seven attention and action during general membership recess in the months of June, July and August are conducted by the District Board of Directors at the discretion and approval of the general membership.

Meeting location for District Seven has for many years been held at the Westward Ho Country Club in Sioux Falls with one exception.

For the past several years at the personal invitation of Theodore Angelos, M.D. (Family Practice), the October meeting has been held at the Canton Country Club. The October meeting includes associated pre-meeting recreational activities of golf and the banquet provided through the generosity of Dr. Angelos includes a pheasant feed served home style and includes all conceivable amenities.

The October meeting includes liaison reports from medical school activities and is a town and gown session of conciliation, fellowship and good will. This meeting has traditionally been extremely well attended, and membership complaints are very few at this session. This generosity, sensitivity, insight and cordiality are matched only by the imposing physical statute of the man who organizes this October activity each year.

Interspersed with the out of town travels to Canton have been the occasional District Seven meeting held in the month of May in Dell Rapids.

This activity, which includes pre-meeting recreational activities of golf at the Dell Rapids Country Club, has been equally as well attended and the gastronomic delights provided for and sponsored by Dr. Paul Aspaas (Family Practice) have been indeed very gratifying and second to none.

Specific events as they transpire are recorded in the monthly minutes of the Seventh District Medical Society general meeting minutes. These are available for perusal by any member in any district of the South Dakota State Medical Association. They will not be reiterated here. Meeting minute copies can be obtained by contacting either Dr. Parry (Pulmonary Medicine) or Dr. Freeman (Neurology).

There are two annual meetings traditionally devoted to presentations from the South Dakota State Medical Association Executive Secretary and the President of the South Dakota State Medical Association. By consensus, these always seem to be highlight meetings for District Seven. District Seven general membership consensus reflects the sentiment that our Executive Secretary, Robert Johnson, continually and consistently demonstrates exemplary conduct in carrying out his duties and responsibilities, and we are reminded of Isaiah 35:1 which states "and the desert shall bloom like a rose."

Since the late 1970's, District Seven membership devotes one regular meeting time to hosting regional State Representatives, Senators and spouses. This event is usually held two to four

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weeks before the annual legislature session commences. It gives everyone an opportunity to share common concerns and to alleviate potential barriers in communication. It is always mutually reassuring to find that we all have similar concerns, hopes and goals even though our paths of realization may differ.

A new but yet unestablished tradition occurred in late Autumn of 1983 when District Seven members hosted an assembly for members of the Sioux Falls Chamber of Commerce.

District Seven membership in 1983 and 1984 has been at peace under the guidance of Dr. Joseph Hamm as the South Dakota State Medical Association President. Dr. Hamm has demonstrated consummate skill and wisdom in discharging his responsibilities.

District Seven members as well as many patients are saddened at the loss of Vernon Cutshall, M.D. (Family Practice) and Charley McDonald, M.D. (Family Practice) who went to their eternal reward in February of 1984.

One has only to visit briefly with any patient who received care from these two physicians to ascertain the respect and admiration that these men engendered. We would all do well to pause and take note of their positive attributes and attempt to emulate them.

District Seven members traditionally eagerly anticipate the cordial manner by which the District Nine members host activities at the annual meeting in Rapid City. Perhaps their close association with the number two industry in the State of South Dakota enables them to hone their skills and espertise in planning and conducting activities.

Respectfully submitted,
Michael Pekas, M.D.
John Ochsner, M.D.
Richard Tschetter, M.D.
Lowell Hyland, M.D.
Richard Gunnarson, M.D.
Councilors, Seventh District

The Reference Committee reviewed the report from the Councilors for the Seventh District Medical Society and recommended it be accepted as submitted.

REPORT OF THE EIGHTH DISTRICT COUNCILOR

The Eighth District Medical Society met three times during 1983-1984. The first meeting was held Wednesday, September 21, 1983, at which time an excellent talk was given by Dr. Eric Rolfsmeyer on "The Advances in Colon and Rectal Surgery." Following this, Doctors Messner and Porter discussed with the group the cost containment program of the Council and also the subcommittee report on neonatal care.

The second meeting was held Wednesday November 30, 1983. Dr. Joseph Hamm, President of the SDSMA, addressed the district members with a timely talk that evoked many thought-provoking questions and answers at the end of Dr. Hamm's presentation.

Robert Johnson, Executive Secretary of the State Medical Association, was also on hand and added much knowledge and insight into the discussion that followed Dr. Hamm's presentation.

During the second meeting, it was recommended that Dr. Richard Honke be nominated for the Community Service Award as a posthumous recognition and that Dr. David Bean be recommended for the Distinguished Service Award.

The third meeting was held on Wednesday, February 29, 1984. Dr. Douglas Gracey of the Mayo Clinic addressed the group with a talk on "The Adult Respiratory Distress Syndrome." A question and answer period followed the excellent presentation.

The nominating committee for the officers for the coming two years presented a slate of officers consisting of Dr. Herbert Soloum of Tyndall as President, Dr. David Smith of Yankton as Vice-President and Treasurer, and Dr. Julie Stevens of Yankton as Secretary.

Also, a ballot was cast for delegates to represent the district at the state medical meeting, and Dr. Morris Radack, Dr. Michael McVay, and Dr. Larry Meyer were selected as delegates to the annual convention. Dr. Ted Sattler, Dr. Duane Reaney, and Dr. David Smith were selected as alternate delegates.

It was also noted that with the selection of Dr. Frank Messner

South Dakota Society Of Pathologists

Officers for 1983-84

Jerry L. Simmons, M.D., President
John F. Barlow, M.D., Vice President
Beth L. Johnson, M.D., Secretary-Treasurer

as Speaker of the House, Dr. Jay Hubner, the alternate councilor, will replace Dr. Messner as a councilor representing the Eighth District. An alternate council member was required, and for this, Dr. John Sternquist was appointed.

Discussion about resolutions to be presented to the state medical meeting ensued. Dr. David Bean asked that a resolution be submitted to voice the support of the State Medical Society for the Human Services Center. Dr. Ted Sattler asked that a resolution be submitted to the effect that the State Medical Association would undertake a program to inform the public about the new diagnosis-related groupings for the patients to better understand what this entails. Both of these resolutions were accepted by the membership of the district society.

Respectfully submitted,
R. I. Porter, M.D.
Councilor, Eighth District

The Reference Committee reviewed the report from the Councilor for the Eighth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE NINTH DISTRICT COUNCILORS

During the past year, the Black Hills District Medical Society has met on six occasions; in May of 1983, September of 1983, November of 1983, December of 1983, February of 1984, and April of 1984. The seventh meeting is scheduled for May, 1984.

James Kullbom, M.D. resigned as President of the Black Hills District Medical Society.

District membership for 1983 totaled 119 active members and 12 honorary members.

Richard Renka, M.D., was elected President; Robert Allen, M.D., was elected Vice-President — President Elect; Nat Whitney, M.D., was elected Secretary-Treasurer; Ed James, M.D., was appointed Councilor. Doctors Tom Krafka and Nat Whitney are Alternate Councilors.

The Black Hills District Medical Society sustained a significant loss not only to our district, but to the entire State Medical Association, on the death of our friend and colleague, Arthur J. Barrett, M.D., in October of 1983. Not only has medicine lost a fine and compassionate practitioner, but Dr. Barrett's driving force of humor and energy, as well as his deep commitment to the State Medical Association, will be sincerely missed. An honorarium has been established.

We also wish to thank Roger Millea, M.D., for so ably representing the Black Hills District Medical Society as a councilor for so many years.

Respectfully submitted,
Robert Ferrell, M.D.
James Jackson, M.D.
Councilors, Ninth District

The Reference Committee reviewed the report from the Councilors for the Ninth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE TENTH DISTRICT COUNCILOR

The Rosebud District Medical Society has nine active members including Dr. Gregg Tobin who joined this year. The official meeting of the district was held in January at which time Dr. Joseph Hamm made his presidential visit along with Robert Johnson, Executive Secretary of the Association. The district met several other times for scientific programs.

Officers elected for 1984 include:

President	Gregg Tobin, M.D.
Vice President	Tony Berg, M.D.
Secretary-Treasurer	R. G. Nemer, M.D.
Delegate	Edwin Sweet, M.D.
Alternate Delegate	Louis Hogrefe, M.D.

Respectfully submitted,
M. George Thompson, D.O.
Councilor, Rosebud District
Medical Society

The Reference Committee reviewed the report from the Councilor for the Tenth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE ELEVENTH DISTRICT COUNCILOR

During the past year, three meetings of the Eleventh District Medical Society were held. At these meetings, business sessions were conducted as needed. Scientific sessions were presented by visiting consultants. At one of the meetings, Robert A. Browne, M.D., who is the Director of Infectious Diseases at Penrose Hospital in Colorado Springs, spoke on "The Update on Management of Medical and Surgical Infections." The annual meeting was held in January of 1984 with Dr. Joseph Hamm, President, and Robert D. Johnson, Executive Secretary, of the South Dakota State Medical Association.

Our annual meeting was held in February, 1984 at which time new officers for the society were elected and are as listed:

President	Ben Henderson, D.O.
Vice President	Jeff Peterson, M.D.
Secretary-Treasurer	Leonard Linde, M.D.
Delegate	Leonard Linde, M.D.
Alternate Delegate	David Yecha, M.D.
Councilor	James F. Wunder, M.D.
Nominating Committee member	Leonard Linde, M.D.

Respectfully submitted,
James F. Wunder, M.D.
Councilor, Eleventh District

The Reference Committee reviewed the report from the Councilor for the Eleventh District Medical Society and recommended it be accepted as submitted.

REPORT OF THE TWELFTH DISTRICT COUNCILOR

During 1983-84, the Whetstone Valley District Medical Society met three times. The first meeting in August was held in Rosholt. Dr. Joseph Hamm, President of the State Medical Association, made his official visit to the district at that time accompanied by Bob Johnson. Current topics of concern to the practicing physician were discussed.

In November, the district met in Milbank, and in March we met in Webster. Both of these meetings included scientific speakers as well as business sessions. District officers were elected at the March meeting for the coming year. These are: President, Vichit Vanadurongvan, M.D.; Secretary-Treasurer, Kanya Vanadurongvan, M.D.

Respectfully submitted,
Joseph Kass, M.D.
Councilor, Twelfth District

The Reference Committee reviewed the report from the Councilor for the Twelfth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

The only meeting of the Commission on Legislation and Governmental Relations was held Wednesday, November 9, 1983, in Sioux Falls.

The Commission reviewed the legislation limiting awards in medical malpractice actions. The law now states that total general damages may not exceed $500,000. This statute becomes sunsetted on June 30, 1986. It was recommended to the Council and the State Medical Association that the licensure of radiologic technologists be opposed.

Punitive damage legislation that was introduced in the 1983 legislature was reviewed. This proposed legislation would have required recovery in a civil action of double cost of defense of a punitive damage claim where the claim was found to be without reasonable basis. The second piece of legislation would have bifurcated the trial of negligence and trial for punitive damages. Further input from the St. Paul Property & Liability Insurance Company and from experience with similar legislation in Florida

was reviewed, and the committee recommended to the Council and State Medical Association that further information be gathered from the AMA office of the general council and a solution be drafted with input from the St. Paul Fire and Marine Insurance Company. It was recommended that no action be taken in the 1984 legislature until such joint solution be drafted.

The Commission's support of child passenger safety legislation was again approved. It was recommended to the Council that the State Medical Association endorse such legislation if introduced into the 1984 session.

The coroner's statute bill was reviewed. Since its defeat in the House State Affairs Committee in 1983, Dr. Brad Randall has continued to work with the coroners and morticians on this matter. It was recommended to the Council that the State Medical Association sponsor this legislation again in the 1984 legislature.

Information on the licensure of athletic trainers was discussed. It was recommended that no action be taken on this matter until specific legislation could be reviewed.

In 1981, the Commission had reviewed actions of the Council not to endorse continued mandatory premarital serology testing. Since this bill was not introduced at this time, it was the recommendation of the Commission to the Council that the State Medical Association endorse the repeal of mandatory premarital serology testing if this legislation was introduced in the 1984 legislative session.

Dr. Don Frost and Dr. Jerry Walton discussed proposed legislation for the licensure of interns and residents by the Board of Medical and Osteopathic Examiners. This licensure would be a special license to practice as a resident physician. It was recommended to the Council and the State Medical Association that this legislation proposal be presented to both the Sioux Falls and the Yankton District Medical Societies for their comments prior to the Executive Committee of the State Medical Association to be held on January 5, 1984.

Bob Johnson indicated that legislation to raise the drinking age to 21 may again be introduced this year. The Commission previously had recommended educational assistance for such legislation only. The Commission recommended at this time to the Council and the State Medical Association that such legislation be supported if it was introduced as legislation.

Information was presented to the Commission on the current status of nurse midwife practice in the state of South Dakota. The position of the American Association of Family Practice was reviewed. It was recommended that no legislation be introduced involving the practice parameters for nurse midwives.

Dr. Tony Javurek presented information on the proposed requirement of cardiopulmonary resuscitation training for high school students. This concept had been approved by the Council on September 23, 1983. The Commission recommended that the State Medical Association co-sponsor this bill in the 1984 legislative session if provisions could be made for students who had physical or other reasons that they could not complete a cardiopulmonary resuscitation course.

Mr. Johnson summarized the summer study of the Legislative Research Council as it related to providing a risk pool for uninsurables and also legislation requiring hospitals to negotiate a cash discount for patients who pay cash for their hospital bill upon discharge.

Dr. Earl Kemp reviewed the current performance of residents in the Family Practice Residency program and the request to the legislature for additional funding in 1984. The Commission recommended to the Council that the State Medical Association continue to endorse adequate funding for the Family Practice Residency program.

The State Health Department had indicated that legislation would be introduced to the 1984 session for the repeal of annual TB testing of school teachers. The Commission recommended to the Council that the State Medical Association endorse the repeal of annual TB testing for teachers, leaving in place the requirement that a TB test be performed upon entering the school system.

Following is a finalized 1984 legislative program as adopted by the Commission:

SPONSORED BILLS
1. Legislation creating an office of the state coroner and amending present coroner's status.
2. Cardiopulmonary resuscitation training for high school students.

ENDORSED BILLS
1. Child passenger safety legislation.
2. Repeal of premarital serology.
3. Legislation to raise the drinking age to 21.
4. Legislation to insure adequate funding of family practice residency program.
5. Repeal of annual TB testing for school teachers.

OPPOSED BILLS
1. Licensure of radiologic technologists.

Respectfully submitted,
James R. Reynolds, M.D., Chairman
Commission on Legislation
and Governmental Relations

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations and recommended acceptance of this report.

REPORT OF THE COMMISSION ON MEDICAL SERVICE

The Commission on Medical Service of the South Dakota State Medical Association held two meetings during the past year; one on October 28, 1983; the second on March 28, 1984.

The Commission has continued to monitor the state disaster plan for South Dakota. Discussion is under way with the subcommittee of the Commission, the Department of Health, and the Medical School for incorporating plans for usage of physicians in case of disasters in the state. A definite plan has not yet been developed.

The Commission continued to evaluate the cost containment report submitted during the previous year. The Commission

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recommended to the Council that the cost containment report become a resource document for the Association, not a policy statement. This was adopted by the Council. It further recommended that the State Medical Association encourage the use of deductibles and co-payments by third party payers, and that the Council review the concept of indemnity insurance, rather than usual and customary. Action on this was delayed by the Council pending the outcome of AMA deliberations on the subject. The Commission also recommended that a series of meetings with business leaders be established concerning cost containment, a recommendation that the Council referred to district medical societies for implementation. At its most recent meeting, the Commission made no further recommendations on the cost containment matter.

At its most recent meeting, the Commission heard a presentation from a representative of American Family Insurance Company concerning letters to policyholders. It was indicated that changes had been made in the letters, and a recommendation was made to the Council to continue to monitor this matter. The Commission further recommended to the Council that Blue Shield of North Dakota be requested to add South Dakota physicians to their Medical Advisory Committee. The Commission also reviewed proposed new HCFA Form 1500, and recommended that no change be made in the form pending clarification of the needs for the proposed changes.

Respectfully submitted,
Loyd R. Wagner, M.D., Chairman
Commission on Medical Service

The Reference Committee reviewed the report of the Commission on Medical Service and recommended acceptance of this report.

REPORT OF THE COMMISSION ON SCIENTIFIC MEDICINE

The Commission met on September 10, 1983, with eleven members and two guests present.

The Commission discussed practice parameters for nurse midwives and recommended that the State Medical Association endorse the position of the American Academy of Family Physicians which calls for the abolishment of nurse midwifery, and that if the nurse midwife law cannot be rescinded, the South Dakota State Medical Association attempt to amend it to require on-site supervision of nurse midwives.

The American College of Emergency Physicians, South Dakota Chapter, requested endorsement of mandatory CPR training for high school students, and the Commission endorsed the concept of requiring basic CPR training in the high school curriculum.

A majority of the Commission's business related to planning the Friday afternoon scientific session during the 1984 annual meeting. Topics to be presented include: 1) new concepts in imaging techniques, 2) new approaches to hypertension, 3) sepsis splenectomy, 4) family abuse, 5) legal implications of reporting abuse and 6) the impaired physician.

Other items of business received after the September meeting were handled through the mail or by the Commission chairman.

Respectfully submitted,
A. J. Janusz, M.D., Chairman
Commission on Scientific Medicine

The Reference Committee reviewed the report of the Commission on Scientific Medicine and recommended acceptance of this report. The Reference Committee commended the Commission members for their work on behalf of the State Medical Association.

REPORT OF THE COMMISSION ON INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON

The Commission met twice during the past year, on August 31, 1983, and at a special meeting held on December 14, 1983.

At the August 31, 1983, meeting, the Commission reviewed an insurance proposal for disability income insurance protection submitted by the Williams Insurance Company. The Commission recommended that the proposal be accepted and endorsed by the State Medical Association for primary or supplemental coverage for our members.

Discussion was then held on the publication of scientific articles from the faculty of the University of South Dakota School of Medicine in the SOUTH DAKOTA JOURNAL OF MEDICINE. It was recommended that the Commission meet with Dr. Quinn, the Dean of the School of Medicine, to discuss this issue further. A special meeting was held on December 14, 1983, with Dr. Quinn in attendance. Dr. Quinn discussed with the Commission the School's request to publish one or two scientific papers per month in the SOUTH DAKOTA JOURNAL OF MEDICINE. He indicated that there was a need on the part of the faculty and residents in training to have access to publication space in the Journal. He indicated that the school would underwrite the cost of these articles and the articles would first be screened by the Medical School and then submitted to the Journal's Editorial Board for final approval prior to publication. Following discussion, it was moved and carried that the Commission recommend to the Council that space in the SOUTH DAKOTA JOURNAL OF MEDICINE be allocated for scientific articles provided by the School of Medicine on a monthly basis, that such articles would be subject to review by the Journal's Editorial Board prior to publication, and that reimbursement to the Journal by the School of Medicine to cover the cost for these publications should be negotiated.

During the past year, the following physicians have died: R. W. Honke, M.D., Wagner; Vernon Cutshall, M.D., Sioux Falls; Howard Wold, M.D., Madison; A. J. Barrett, M.D., Rapid City; C. J. McDonald, M.D., Sioux Falls; E. A. Rudolph, M.D., California; and Robert Donahoe, M.D., Orlando, Florida.

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Health Career Grant Fund reported the following activities during the past twelve months:

Balance in Savings Account 03-01-83		$ 7,343.75
Income		
Interest	$ 615.93	
Principal	1,067.99	
	$ 1,683.92	1,683.92
		$ 9,027.67
Expenses		
Transferred to C.D.	$ 5,000.00	
Four Grants @ $500	2,000.00	
	$ 7,000.00	7,000.00
		$ 2,027.67
Balance in Savings Account 02-29-84		$ 2,027.67
Certificates of Deposit		
#1423	$ 7,438.64	
02-28-85 — 1 year — 9.40%		
#553	15,640.31	
07-09-84 — 6 months — 9%		
#1266	5,000.00	
12-09-85 — 2½ years — 9.25%		
Value 02-20-84	$28,078.95	$28,078.95
. Interest earned on C.D.'s — C.D. #1045 cashed 02-28-84 and 1-year certificate purchased #1423		
#553	$ 1,290.01	
#1045	1,808.74	
	$ 3,098.75	
Assets 02-29-84		
Savings Account	$ 2,027.67	
Certificates of Deposit	28,078.95	
Outstanding Loans	6,530.57	
	$36,637.19	$36,637.19

During the year, the Commission has reviewed each financial report of the State Medical Association, the general account and the building fund. The Budget and Audit Committee, consisting of the Executive Commission and Chairman of this Commission, considered and reviewed a budget for the fiscal year 1984-85, and it was submitted to the Council for its consideration and transmitted to the House of Delegates. The proposed budget is attached as part of this report.

Respectfully submitted,
Jay W. Hubner, M.D., Chairman
Commission on Internal Affairs,
Communications & Liaison

The Reference Committee reviewed the report of the Commission on Internal Affairs, Communications and Liaison and recommended acceptance of this report. The Reference Committee commended the Commission members for their work on behalf of the State Medical Association.

1984-85 BUDGET
SOUTH DAKOTA STATE MEDICAL ASSOCIATION

GENERAL FUND
INCOME

ITEM	BUDGETED 83-84	PROPOSED 84-85
State Dues	$183,000.00	$190,000.00
Annual Meeting	25,000.00	25,000.00
Refunds	5,500.00	5,500.00
Car Reimbursement	500.00	500.00
Continuing Med. Education	750.00	1,000.00
Salary Reimbursement	12,900.00	18,900.00
Other Programs		
7th Dist. Salary Reimb.	1,350.00	1,350.00
Equip. Repl. Fund	2,400.00	2,400.00
AAFP Salary Reimb.	1,500.00	1,500.00
Medical Student Dues	500.00	500.00
Collection Service	4,000.00	3,500.00
Interest	17,000.00	25,000.00
Ret. Lives Res. & Other	3,500.00	4,000.00
	$257,900.00	$279,150.00

EXPENSES

ITEM		
Salaries	$114,000.00	$130,000.00
Social Security	7,000.00	7,500.00
Legal & Audit	7,500.00	7,500.00
Telephone	4,000.00	3,000.00
Office Supplies	9,000.00	10,000.00
Dues & Subscriptions	900.00	900.00
Physicians' Travel	14,000.00	14,000.00
Annual Meeting	18,000.00	20,000.00
Public Relations	6,500.00	7,000.00
Journal Subsidy	6,000.00	9,000.00
Postage	7,000.00	7,000.00
Miscellaneous	100.00	100.00
Legislation	6,000.00	5,500.00
Staff Travel	12,000.00	12,000.00
Insurance	3,000.00	3,000.00
Retirement/Fringe	25,000.00	30,000.00
Car Operation & Maint.	3,500.00	3,000.00
Auxiliary Newsletter	1,300.00	1,600.00
Employment Tax	800.00	800.00
Continuing Med. Education	750.00	750.00
Income Tax	750.00	300.00
Medical Student Fund	500.00	500.00
	$247,600.00	$273,450.00
Reserve	10,300.00	5,700.00
	$257,900.00	$279,150.00

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JOURNAL INCOME

ITEM	BUDGETED 83-84	PROPOSED 84-85
Advertising	$18,500.00	$17,000.00
Subscription	1,200.00	1,200.00
Refunds	720.00	720.00
Journal Subsidy	6,000.00	9,000.00
Miscellaneous	600.00	600.00
	$27,020.00	$28,520.00

EXPENSES

ITEM	BUDGETED 83-84	PROPOSED 84-85
Salaries	$ 2,200.00	$ 2,200.00
Legal & Audit	100.00	100.00
Social Security	100.00	50.00
Telephone	115.00	115.00
Postage	2,000.00	1,800.00
Office Supp. & Print.	22,505.00	24,255.00
	$27,020.00	$28,520.00

BUILDING FUND INCOME

ITEM	BUDGETED 83-84	PROPOSED 84-85
Foundation Rent	$14,740.00	$15,640.00
Board of Exam. Rent	4,400.00	4,800.00
Interest Income	11,000.00	12,000.00
	$30,140.00	$32,440.00

EXPENSES

ITEM	BUDGETED 83-84	PROPOSED 84-85
Salaries	$16,140.00	$17,940.00
Legal & Audit	1,500.00	1,500.00
Social Security	5,000.00	5,000.00
Utilities	3,500.00	4,000.00
Maint. & Supplies	4,000.00	4,000.00
	$30,140.00	$32,440.00

REPORT OF THE COMMISSION ON PROFESSIONAL LIABILITY

This past year the Commission met on one occasion in Sioux Falls on October 27, 1983. During this meeting, the Commission reviewed various types of legislation that have been instituted around the nation regarding professional liability, and also reviewed the proposals of increases in liability premiums across the nation. The majority of the time was spent discussing and planning a follow-up Risk Management session to the 1983 program. The Council had requested that the Commission provide approximately six hours of additional follow-up meeting on Rick Management at the 1984 annual meeting. During this session, it was decided to attempt to have a program based on a "mock trial or deposition taking" provided by individuals with experience in the medical-legal field. We hope that this follow-up of a Risk Management seminar will continue to be of value to the practicing physicians in South Dakota and be as well accepted as the 1983 annual meeting.

Respectfully submitted,
M. L. Radack, M.D., Chairman
Commission on Professional Liability

The Reference Committee reviewed the report of the Commission on Professional Liability and recommended acceptance of this report. The Reference Committee commended the Commission members for their work on behalf of the State Medical Association.

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REPORT OF THE COMMITTEE FOR CONTINUING MEDICAL EDUCATION

This year the Committee for Continuing Medical Education will be holding a meeting in conjunction with the State Medical Association Meeting in mid-April 1984. Our plan is to update our present policies of review and approval for continuing medical education and to prepare for an anticipated inspection of our program.

Respectfully submitted,
K. G. Koob, M.D., Chairman
Committee for Continuing Medical Education

The Reference Committee reviewed the report of the Committee for Continuing Medical Education and recommended acceptance of this report.

REPORT OF THE GRIEVANCE COMMISSION

The Grievance Commission of the State Association has functioned in its usual fashion during the 1983-84 year without any unusual incidents.

It is noted that there have been markedly less serious complaints from consumers during the past year. Hopefully, this indicates not only a continuation of the generally fine care that patients receive in this state, but also even greater compassion for the complaints of the patient.

Also, during the past year there seems to be a great deal fewer complaints from patients which were instigated by undue criticism of practitioners by fellow physicians.

I would like to take this opportunity to thank the other commission members, Dr. Russell Harris of Rapid City, Dr. Winston Odland of Aberdeen, Dr. Durward Lang of Sioux Falls, and Dr. Bruce Lushbough of Brookings for their continued support and work.

Respectfully submitted,
Duane B. Reaney, M.D., Chairman
Grievance Commission

The Reference Committee reviewed the report of the Grievance Commission and recommended acceptance of this report.

REPORT OF THE LONG RANGE PLANNING COMMITTEE

The Long Range Planning Committee held teleconferences on September 14, 1983, and April 3, 1984. We finalized the membership survey questionnaire at the fall meeting. The survey was conducted with a 53% response from our Association members. We discussed the results of the survey at the April meeting with future plans to review indepth five areas. These are the SOUTH DAKOTA JOURNAL OF MEDICINE, membership benefits, annual meeting format, continuing medical education, and public relations program. The plan is to have recommendations of each of these areas for the Fall, 1984, Council meeting.

Respectfully submitted,
Gerald D. Loos, M.D., Chairman
Long Range Planning Committee

The Reference Committee reviewed the report of the Long Range Planning Committee and recommended acceptance of this report.

REPORT OF THE LIAISON COMMITTEE

The Liaison Committee between the South Dakota State Medical Association and the South Dakota State Health Department had only a single meeting this past year which was held October 13, 1983. At this meeting several items were discussed including a re-designed universal claim form for physician services, the rules and use of generic drugs that are approved by the State Health Department, incidents of possible quackery within the state, and also several items of interest that will be handled in the legislature.

The meeting was felt to be informative and instructive to persons in both the Medical Association and State Health Department.

Members of the committee are to be commended for their useful input and for the time expended on behalf of the Medical Association. These reports have also been submitted and reviewed at appropriate council meetings.

We will plan continued exchanges with the State Health Department, hopefully for the betterment of both the Medical Association and the people of South Dakota.

Respectfully submitted,
Frank D. Messner, M.D., Chairman
Liaison Committee

The Reference Committee reviewed the report of the Liaison Committee and recommended acceptance of this report.

REPORT OF THE SOUTH DAKOTA POLITICAL ACTION COMMITTEE

The Board of Directors of the South Dakota Political Action Committee met February 1, 1983; June 3, 1983; and January 23, 1984. At the February, 1983, meeting, we adopted a revision of our Constitution and Bylaws, providing that each district shall have one physician member and one Auxiliary member for each potential fifty members. At the meeting of the Council on June 2, 1983, the current Board of Directors was elected to serve a three year term, until the annual meeting in June, 1986, and at the June 3 annual SoDaPAC meeting, I was elected to a two year term as Chairman.

As of April, 1984, the members of the Board of Directors are W. R. Taylor, M.D., Harvey Hart, M.D., Mrs. Marie Hovland, T. J. Wrage, M.D., Curtis Wait, M.D., Mrs. Barbara Wait, John Davis, M.D., Robert Hohm, M.D., Michael Haley, M.D., Mrs. Boots Mabee, Courtney Anderson, M.D., Mr. Les Kinstad, Karl Wegner, M.D., R. I. Porter, M.D., Mrs. Marlys Porter, N. R. Whitney, M.D., Mrs. Mary Ann Harris, Robert Bloemendaal, M.D., Raymond Nemer, M.D., Michael Pekas, M.D., James Wunder, M.D., James Ryan, M.D., Ellen Kunz, Duane Reaney, M.D., Kay Reaney, and L. F. Nelson, M.D. The following positions (as of March) still need to be filled: Districts 1, 2, 4, 5, 10, 11, and 12 — one Auxiliary member; District 7 — one physician member and five Auxiliary members; District 9 — one physician member to complete Dr. Barrett's term, and one Auxiliary member.

The Board members share the sorrow of the entire South Dakota medical community in the death of Director Arthur J. Barrett, M.D., of Rapid City.

The Board members welcome our new lobbyist Lorin Pankratz, who has joined Bob Johnson in working with SoDaPAC from the Association office. Lorin fills the vacancy left by Kevin Loge, who is now Blue Shield Professional Relations Representative.

In June, 1983, the Huron District Medical Society donated $100 to the Education Fund. We want to express our appreciation to the Huron District, and we urge all districts to contribute to the Educational Fund.

At the annual meeting in June, 1984, we will be reviewing all election contests within the state. This is a critical year for our state and federal governments, since we will have to re-elect or replace both Senator Pressler and Congressman Daschle, as well as all of our 35 State Senators and 70 State Representatives. The Statehouse is especially important this year, since we are changing to one senator, two representative districts, and so in many districts we will be voting for new people.

How effective is SoDaPAC? By my criteria of measurement, it is very effective. Our aim is to support the campaigns of the candidates whom we believe to be in agreement with us, and in the 1982 elections, we supported 66 candidates for our state legislature. Of these, 56 or 85% were elected. I think the bottom line is the Legislative Report in the Grab Bag — most of the bills which the South Dakota Medical Association either sponsored or endorsed were passed — most of the bills which we opposed were either defeated or amended to remove their objectionable features.

The year 1984 is a new election and, therefore, a completely new situation. The more members we have in SoDaPAC, the more effective we can be in maintaining environments favorable to medicine in both Washington and Pierre.

Respectfully submitted,
Nathaniel R. Whitney, M.D.
Chairman, SoDaPAC

The Reference Committee reviewed the report of the South Dakota Political Action Committee and recommended acceptance of this report. In this important election year of 1984 the Reference Committee recommended that the House of Delegates encourage active participation in the political process by physician and spouse membership in SoDaPAC.

REPORT OF THE BOARD OF DIRECTORS OF THE SOUTH DAKOTA MEDICAL SCHOOL ENDOWMENT ASSOCIATION

The Board of Directors of the South Dakota Medical School Endowment Association met as prescribed by the Association Bylaws during the Annual Meeting of the South Dakota State Medical Association, which was held in Sioux Falls, June 2-5, 1983. Throughout the year, business has been conducted by telephone or mail through the coordination of the staff of the Medical Association.

The goals of the Endowment Association continue to be the support of deserving medical students of the University of South Dakota School of Medicine through loans made at reasonable rates of interest and support of the office of the Dean of the school in essential activities for which there are no other funds. During 1983, loans were made in the amount of $20,000, and already in 1984, the Endowment has granted loans totaling $26,500. The Endowment Association was left a share of an estate with stipulations that the interest which accrued be given to certain qualified students at the University of South Dakota School of Medicine. In 1983, the first grants were awarded to four students totaling $3,700.

Contributions and memorials for 1983 totaled $21,216 from 136 donors. Contributions to the Endowment are encouraged throughout the year with special solicitations in the summer and autumn. The practicing physicians of South Dakota continue to be the major contributors. The Endowment Association remains an essential function of the South Dakota State Medical Association.

Respectfully submitted,
The Board of Directors
Joseph N. Hamm, M.D., Chairman
Robert R. Giebink, M.D.
Warren Jones, M.D.
Bruce C. Lushbough, M.D.
Gerald E. Tracy, M.D.
Bruce H. Allen, M.D.
Theodore H. Sattler, M.D.

The Reference Committee reviewed the report of the Board of Directors of the South Dakota Medical School Endowment Association and recommended acceptance of this report.

REPORT OF THE PRESIDENT OF THE SOUTH DAKOTA MEDICAL SCHOOL ENDOWMENT ASSOCIATION

The President has attended the meetings of the South Dakota Medical School Endowment Association throughout the year. He wishes to thank the Board of Directors and the physicians of South Dakota who have given support to medical education through the Endowment Association during the past year.

Respectfully submitted,
Joseph N. Hamm, M.D., President
South Dakota Medical School
Endowment Association

The Reference Committee reviewed the report of the President of the South Dakota Medical School Endowment Association and recommended acceptance of this report.

REPORT OF THE ARCHIVES AND HISTORY COMMISSION

The Commission did not meet during the past year; however, historical records continue to be gathered at the executive office. A contract between the South Dakota State Medical Association and the Center for Western Studies at Augustana College, Sioux Falls, provides for the storage and maintenance of the Association's records. In the fall of 1983 the first collection of these records was transferred to the Center for Western Studies.

Unfortunately in February Dr. Charles McDonald, Chairman of this Commission, died. His interest in the preservation of the history of South Dakota and the South Dakota State Medical Association was instrumental in establishing this Commission and in arranging for permanent record storage at the Center for Western Studies.

Respectfully submitted,
C. B. McVay, M.D.
David Buchanan, M.D.
Virginia Tracy, M.D.
Roscoe Dean, M.D.
Archives and History Commission

The Reference Committee reviewed the report of the Archives and History Commission and recommended acceptance of this report.

PRESIDENTIAL OATH OF OFFICE

I SOLEMNLY SWEAR THAT I shall carry out the duties of the President of the South Dakota State Medical Association to the best of my ability. I shall strive constantly to maintain the ethics of the medical profession and to promote the public health and welfare. I shall dedicate myself and my office to improving health standards and to the task of bringing increasingly improved medical care to the people of South Dakota. I shall uphold the Constitution and Bylaws of the AMA and the South Dakota State Medical Association. I shall champion the cause of freedom in medical practice and freedom for all my fellow Americans.

I do solemnly swear that I will discharge the duties of this office to the best of my ability, so help me God.

MINUTES OF SOUTH DAKOTA MEDICAL SERVICE, INC. CORPORATE BODY MEETING

Friday, 9:30 a.m. — Howard Johnson's Motor Lodge
June 8, 1984 — Rapid City, South Dakota

Chairman Ortmeier called the meeting of the Corporate Body of South Dakota Medical Service, Inc., to order at 9:30 a.m. on Friday, June 8, 1984, at Howard Johnson's in Rapid City, South Dakota.

Upon roll call, the following members of the Corporate Body of the South Dakota Medical Service, Inc., were present:

Drs. Joseph Hamm, Howard Saylor, Jr., Richard Gere, William O. Rossing, Frank Messner, Gerald E. Tracy, Bruce Lushbough, Durward Lang, Jerome A. Eckrich, Jr., James Larson, Curtis Wait, R. Curtis Jahraus, David Buchanan, Charles D. Monson, Michael Pekas, Denny Ortmeier, Richard Gunnarson, Lowell Hyland, Richard Porter, Jay Hubner, Tom Krafka, Robert L. Ferrell, James Jackson, M. George Thompson, Joseph Kass, William R. Taylor, Winston Odland, Parry Nelson, W. Nicol Guddal, Richard Holm, Tad Jacobs, Ravi Kapur, William Huet, Charles Flohr, Gary VanErt, James Ryan, A. J. Javurek, George Bruins, Dorrance Ensburg, Jerry Simmons, James Quale, David Hoversten, Bradley Randall, Rodney Parry, Jeffrey B. Hagen, Jerome W. Freeman, Guy Tam, Morris Radack, Larry Meyer, Richard Renka, David Sandvik, Nathaniel Whitney, Charles Gwinn, Edwin P. Sweet, Leonard Linde and Edward A. Johnson.

A quorum being present, the Chairman declared the annual meeting of the Membership of the Corporate Body of South

Dakota Medical Service, Inc., to be duly in session for the transaction of business.

Dr. Joseph Hamm moved that reading of the minutes of the last meeting of the Corporate Body, being the 1983 Annual Meeting, be waived, the same having been published in the Handbook and previously mailed to each member. Such motion was seconded by Dr. Howard Saylor, Jr. Upon voice vote, the same was approved unanimously.

Chairman Ortmeier referred the delegates to the Chairman's message in the Delegates' Handbook and stated that he would only point out a few of the highlights. He noted that in 1981 and 1982 Blue Shield had underwriting losses totaling $1,450,000. Fortunately, 1983 was a turn around year and Blue Shield showed an underwriting gain of $750,000. In 1981 Blue Shield's gross premium income was $13,750,000 as compared to $19,350,000 in 1983. He noted that in spite of a large claims increase, Blue Shield lowered its administrative expenses by decreasing management staff with no extra time occurring on claim payments.

In addition to the foregoing, Blue Shield was instrumental in offering new products to the public with Medigap and Medigap Plus Certificates a major part thereof. Chairman Ortmeier said that a great deal of credit is due to the excellent working relationship between Blue Shield and the physician community and the cooperation the South Dakota physicians have shown towards Blue Shield.

In conclusion he stated that a great deal of credit should be given to Blue Shield's Board of Directors, and its staff. He personally thanked James Jelbert for twelve years of devoted service to Blue Shield and the financial expertise he brought to the Board which helped Blue Shield to weather a very poor economic environment in 1982 and prior years.

The Chairman called for President Ben Johnson to present the Financial Report.

Mr. Johnson referred the delegates to the written 1983 Annual Report which was sent to them along with their Delegates' Handbook. He noted that Blue Shield had a record year with a net gain to surplus of $1,400,000. The 1983 year end unassigned surplus of Blue Shield in the sum of $3,275,000 amounts to approximately 1.8 months of administrative and claims expense. Dr. James Larson moved approval of the Financial Report. Such motion was seconded by Dr. R. Curtis Jahraus. Upon vote, the same was approved unanimously.

The Chairman called for the report of the Board of Directors Nominating Committee.

Dr. John Stransky, Chairman of the Blue Shield Board Nominating Committee, stated that the directors whose term of office expired this year were Roscoe Dean, M.D., Robert Bloemendaal, M.D., and James Jelbert. He reported that Drs. Dean and Bloemendaal were eligible for reelection to the Board and that James Jelbert of Spearfish was not eligible for reelection by reason of having served four consecutive terms, for a twelve year period, on the Blue Shield Board of Directors. Dr. Stransky stated that the Nominating Committee recommended the reelection of Roscoe Dean, M.D., and Robert Bloemendaal, M.D., to the Blue Shield Board of Directors and the election of James Dunn of Lead, South Dakota, for election to the Board of Directors.

The Chairman called for nominations from the floor.

Dr. Durward Lang moved that the report of the Nominating Committee be accepted and the Secretary be instructed to cast a unanimous ballot for the nominees, namely, Drs. Dean and Bloemendaal and James Dunn. The foregoing motion was seconded by Dr. Sattler. Upon voice vote, the motion was approved unanimously.

The Chairman asked if there was any other old business which any delegate desired the body to consider. No old business was presented by any delegate.

The Chairman called for consideration of any new business that any delegate would like to bring before the corporate body. No new business was presented by any delegate.

The Chairman asked for James Jelbert to come forward. Mr. Jelbert came forward and the Chairman asked the delegates to recognize Mr. Jelbert's twelve years of service to South Dakota Medical Service, Inc., Blue Shield, and presented him with a wall plaque noting his twelve years of outstanding service to Blue Shield. James Jelbert was recognized by the delegates with a round of applause.

The Chairman asked if there was any further business any one cared to present to the Body. There being none, Durward Lang moved adjournment of the Corporate Body of South Dakota Medical Service, Inc. The motion was seconded by Dr. Saylor. Upon voice vote, the same was approved unanimously.

The meeting adjourned at 10:00 a.m.

John H. Zimmer
Secretary

DISTINGUISHED SERVICE AWARD

Started in 1951 — T. F. Riggs, M.D., Pierre (deceased)
1952 — H. Russell Brown, M.D., Watertown (deceased)
1953 — Guy Van Demark, M.D., Sioux Falls (deceased)
1954 — J. C. Ohlmacher, M.D., Vermillion (deceased)
1955 — R. G. Mayer, M.D., Aberdeen (deceased)
1956 — J. C. Ohlmacher, M.D., Vermillion (deceased)
1957 — W. E. Donahoe, M.D., Sioux Falls (deceased)
1958 — Drs. J. C. Hagin (deceased), M. W. Pangburn (deceased), and James DeGeest, Miller
1958 — J. F. Brenckle, M.D., Superior, Wisc. (deceased)
1958 — Mrs. Agnes Holdridge, Madison
1959 — Walter L. Hard, Ph.D., Vermillion
1959 — Rev. and Mrs. Robert O. Bates, Sturgis
1959 — R. M. Kilgard, M.D., Watertown (deceased)
1960 — L. J. Pankow, M.D., Sioux Falls (deceased)
1961 — Gregg M. Evans, Ph.D., Custer
1962 — Edward Shaw, Ph.D., Vermillion (deceased)
1963 — Arthur A. Lampert, M.D., Rapid City
1964 — John C. Foster, Phoenix, Arizona
1965 — A. P. Reding, M.D., Marion
1966 — Mrs. C. Rodney Stoltz, Watertown
1967 — Mrs. William Fish, Watertown
1968 — G. J. Bloemendaal, M.D., Ipswich
1969 — F. W. Haas, M.D., Yankton (deceased)
1970 — Paul Bunker, M.D., Aberdeen (deceased)
1971 — E. T. Lietzke, M.D., Beresford (deceased)
1972 — C. B. McVay, M.D., Yankton
1973 — G. E. Tracy, M.D., Watertown
1974 — J. A. Muggly, M.D., Madison (deceased)
1975 — Harvey Wollman, Hitchcock
1976 — R. H. Quinn, M.D., Sioux Falls
1977 — E. H. Heinrichs, M.D., Vermillion
1978 — John Olson, Sioux Falls, and Evans Nord, Sioux Falls
1979 — Helen Jane Hare, M.D., Rapid City

1980 — Warren Jones, M.D., Sioux Falls
1981 — Saul Friefeld, M.D., Brookings
1982 — G. Robert Bartron, M.D., Watertown
1983 — Oscar J. Mabee, M.D., Mitchell
1984 — Karl Wegner, M.D., Sioux Falls

COMMUNITY SERVICE AWARD

1961 — R. A. Buchanan, M.D., Huron (deceased)
1962 — Roland F. Hubner, M.D., Yankton
1963 — George W. Mills, M.D., Wall (deceased)
1964 — John C. Hagin, M.D. Miller (deceased)
1965 — Alonzo P. Peeke, M.D., Volga
1966 — Hugo C. Andre, M.D., Vermillion (deceased)
1967 — G. Robert Bartron, M.D., Watertown
1968 — M. M. Morrissey, M.D., Pierre (deceased)
1969 — N. J. Sundet, M.D., Kadoka (deceased)
1970 — W. H. Saxton, M.D., Huron (deceased)
1971 — R. E. Van Demark, M.D., Sioux Falls
1972 — R. H. Hayes, M.D. Wall
1973 — B. F. King, M.D., Aberdeen (deceased)
1974 — M. C. Tank, M.D. Brookings
1975 — Karl Wegner, M.D., Sioux Falls
1976 — John T. Elston, M.D., Rapid City
1977 — W. F. Stanage, M.D., Pierre
1978 — C. S. Roberts, Jr., M.D., Brookings
1979 — C. J. McDonald, M.D., Sioux Falls
1980 — E. A. Johnson, M.D., Milbank
1981 — J. A. Muggly, M.D., Madison (deceased)
1982 — Robert R. Giebink, M.D., Sioux Falls
1983 — Theodore H. Sattler, M.D., Yankton
1984 — Paul Hohm, M.D., Huron

AESCULAPIUS AWARD

1966 — Paul R. Leon, M.D.
Walter Miller, M.D., Aberdeen
1968 — H. Phil Gross, M.D., Sioux Falls

FIFTY YEAR CLUB MEMBERS

C. V. Auld, Plankinton (deceased)
G. J. Bloemendaal, M.D., Ipswich
W. C. Brinkman, M.D., Sisseton (deceased)
R. A. Buchanan, M.D., Huron (deceased)
John L. Calene, M.D., California (deceased)
Myrtle Carney, M.D., Ft. Worth, Texas
J. C. Clark, M.D., Sioux Falls (deceased)
F. L. Class, M.D., Huron (deceased)
M. E. Cogswell, M.D., Wolsey (deceased)
J. Cook, M.D., Bonesteel (deceased)
G. I. W. Cottam, M.D., Sioux Falls
Harold L. Crane, M.D., Avon, Conn. (deceased)
S. A. Donahoe, M.D., Sioux Falls (deceased)
W. E. Donahoe, M.D. Sioux Falls (deceased)
J. A. Eckrich, Sr., M.D., Aberdeen
V. W. Embree, M.D., Pierre (deceased)
W. D. Farrell, M.D., Aberdeen (deceased)
R. B. Fleeger, M.D., Lead (deceased)
R. R. Fisk, M.D., Flandreau (deceased)
F. W. Freyberg, M.D., Mitchell (deceased)
E. E. Gage, M.D., Sioux Falls (deceased)
D. A. Gregory, M.D., Glasgow, Mont.
E. H. Grove, M.D., Arlington (deceased)
J. C. Hagin, M.D., Miller (deceased)
Lyle Hare, M.D., Spearfish (deceased)
John F. Hill, M.D., Yankton (deceased)
J. A. Hohf, M.D., Yankton (deceased)
F. S. Howe, M.D., Deadwood (deceased)
A. H. Hovne, M.D., Salem (deceased)
Roland Hubner, M.D., Yankton
A. S. Jackson, M.D., Rapid City (deceased)
R. J. Jackson, M.D., Hot Springs (deceased)
J. A. Jacotel, M.D., Milbank (deceased)
G. T. Jordan, M.D., Vermillion (deceased)
F. F. Keene, M.D., Wessington Springs (deceased)
Ray Lemley, M.D., Rapid City (deceased)
J. H. Lloyd, M.D., Mitchell
O. J. Mabee, M.D., Mitchell
P. V. McCarthy, M.D., Aberdeen (deceased)
G. W. Mills, M.D., Wall (deceased)
B. C. Murdy, M.D., Aberdeen (deceased)
T. F. O'Toole, M.D., Rapid City (deceased)
N. T. Owen, M.D., Rapid City (deceased)
L. L. Parke, M.D., Canton (deceased)
C. C. Pascale, D.O., Centerville
A. P. Peeke, M.D., Volga
M. O. Pemberton, M.D., Deadwood (deceased)
R. J. Quinn, M.D., Sioux Falls (deceased)
F. J. Radusch, M.D., California (deceased)
T. B. Ranney, M.D., Aberdeen (deceased)
T. F. Riggs, M.D., Pierre (deceased)
I. R. Salladay, M.D., Ft. Meade (deceased)
W. H. Saxton, M.D., Huron (deceased)
H. L. Saylor, M.D., Huron (deceased)
C. E. Sherwood, M.D., Brookings (deceased)
Arthur W. Spiry, M.D., Mobridge
Myron Tank, M.D., Brookings
F. J. Tobin, M.D., Mitchell (deceased)
Leonard W. Tobin, M.D., Mitchell
J. S. Tschetter, M.D., Huron (deceased)
Paul Tschetter, M.D., Huron
F. W. Valkenaar, M.D., Chancellor (deceased)
G. E. VanDemark, M.D., Sioux Falls (deceased)
H. P. Volin, M.D., Lennox (deceased)
C. H. Weishaar, M.D., Aberdeen (deceased)
J. R. Westaby, M.D., Madison (deceased)
G. E. Zimmerman, M.D., Missoula, Montana (deceased)

C. B. ALFORD AWARD

1974 — Roscoe Dean, M.D., Wessington Springs
1975 — Gerald Tracy, M.D., Watertown
1976 — Robert Westaby, M.D., Hot Springs
1977 — Robert VanDemark, M.D., Sioux Falls
1978 — Howard Saylor, M.D., Huron
1979 — J. D. Bailey, M.D., Rapid City

1980 — John T. Elston, M.D., Rapid City
1981 — T. H. Sattler, M.D., Yankton
1982 — Bedford T. Otey, M.D., Flandreau
1983 — Robert H. Quinn, M.D., Sioux Falls
1984 — Granville Steele, M.D., Aberdeen

Letters To The Editor

Dear Sir:

Please extend my sincere thanks to the South Dakota State Medical Association for the financial award I recently received. Your support is very much appreciated.

Sincerely,
Mica Newman-Koehn
Sioux Falls, SD

Dear Mr. Johnson,

I sincerely thank you for the South Dakota State Medical Association Award. This award will help lessen the burden of the high costs of medical school. I will do my best to live up to the expectations of the South Dakota State Medical Association and I will see that this generous award is used wisely. I have every intention of repaying this award when I become financially able. Once again, I thank you.

Sincerely,
Colin K. Ohrt
Minneapolis, MN

Dear Sirs:

I would like to thank the South Dakota State Medical Association for awarding me a $500 scholarship. It will be useful in helping me pay for my third year in medical school. I plan on using the money to pay part of my tuition. Once again, I thank you for contributing to the education of medical students such as myself in South Dakota.

Sincerely,
Bruce C. Gehrke
Brookings, SD

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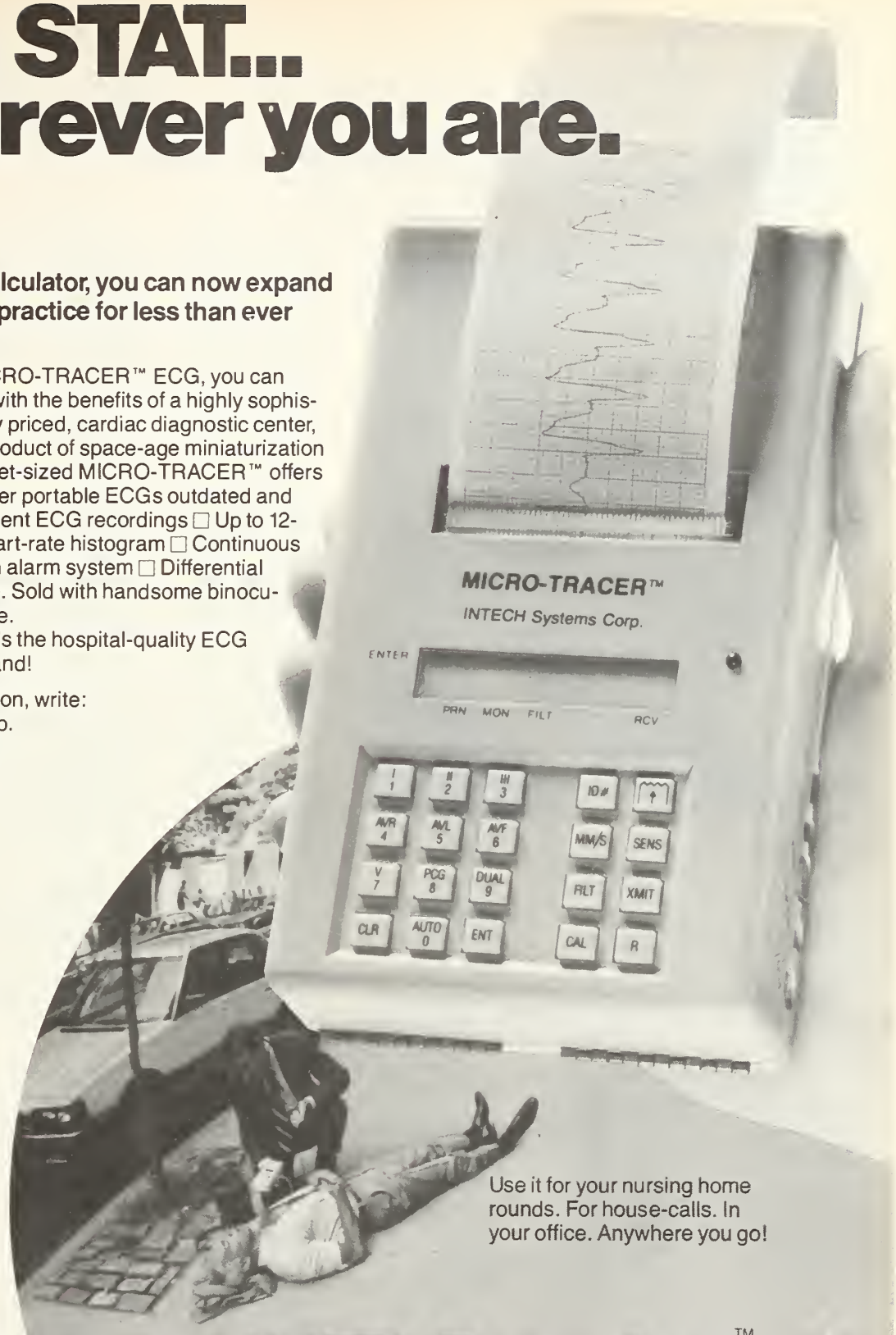

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1984 SOUTH DAKOTA FAMILY DOCTOR OF THE YEAR

Bruce C. Lushbough, M.D. of Brookings, South Dakota is 1984 South Dakota Family Doctor of the Year.

Dr. Lushbough has been an active member of the South Dakota Academy of Family Physicians since 1964. He has been actively engaged as a family physician with the Brookings Clinic since 1961, having been in active practice since that time. Dr. Lushbough is a Diplomate of the American Board of Family Practice, 1972, recertified in 1978. He holds an unrestricted license to practice medicine in South Dakota and is an active member of the Brookings Community Hospital medical staff.

While born in Crawford, Nebraska in 1933, Dr. Lushbough was raised in the Black Hills of South Dakota where his parents owned and operated a hardware store in Sturgis. His mother has been South Dakota Mother of the Year. Bruce was an outstanding student and athlete during his high school days, being a starting regular on a State Class A championship basketball team. He attended the University of South Dakota for his undergraduate and basic science medicine. While there he met his wife, South Dakota native Ila Larson of Rosholt. Bruce received his M.D. degree in 1958 from Jefferson Medical College in Philadelphia where he was elected to Alpha Omega Alpha Honor Medical Society. After his internship at St. Luke's Hospital in Duluth, Minnesota and two years as Lieutenant with the U.S. Navy Medical Corps, Bruce entered practice with the Brookings Clinic. He is still in practice with his original group.

Dr. Lushbough has been a member of the South Dakota State Medical Association since 1961. He served as Chairman of the Scientific Commission, and has been a member and Chairman of the Council. He served as President of SDSMA 1981-82, his wife Ila serving as President of the SDSMA Auxiliary during the same year. He was named an Alternate Delegate to the AMA in 1983.

Bruce received his initial appointment as Preceptor with the University of South Dakota School of Medicine in 1971. He was named Clinical Associate Professor in the Department of Family Medicine, USDSM, in 1976, and promoted to Clinical Professor in 1982. He received the Edward J. Batt Award in 1981, being named Outstanding Faculty Member, Department of Family Medicine, USDSM. He is also Adjunct Professor of Sports Medicine, South Dakota State University, appointed to that faculty position in 1976.

Dr. Lushbough's Academy membership started in 1964. He is a former Mental Health Committee Chairman of SDAFP and served as Vice President and President-Elect leading to his presidential year 1973-74. He was elected Delegate-Elect, SDAFP, in August 1983 and his first two year term began January 1984.

Dr. Lushbough's service to his community has been outstanding. In addition to his medical student teaching, State Medical Association, and Academy activities, he has been team physician at SDSU for nearly 20 years. In this capacity he has served with distinction and has been singularly honored by several Jackrabbit athletic teams. His interest in sports medicine has been shared with others by being a well accepted speaker on a variety of sports medicine topics, especially as they apply to the college level athlete. Dr. Lushbough has also served his community as a member of the Brookings School Board. During Dr. Lushbough's time in Brookings, he has seen the construction of a new Brookings Clinic and a new Brookings Community Hospital, both institutions benefitting from his input and counsel.

Bruce and Ila have been active in their church in addition to raising two children. Son Bruce C., Jr. is a geographer, and daughter Kathryn is a medical student at USDSM.

South Dakota State Medical Association Roster — 1984
Membership by Districts

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Anderson, Esther E. ..... Aberdeen
Bachmayer, Jay D. ..... Aberdeen
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Berg, Sterling ..... Redfield
*Bloemendaal, Gerrit J. .... Ipswich
Brevik, Alan K. ..... Redfield
Broadhurst, Kennon E. ... Aberdeen
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Carter, Peter B. ..... Aberdeen
Chang, Joe P. ..... Aberdeen
Chavier, Juan R. ..... Aberdeen
Chen, Cheng-Fu ..... Aberdeen
Christopher, John R. ..... Aberdeen
Colberg, Douglas ..... Aberdeen
D'Souza, Edward P. ..... Aberdeen
Eckrich, Jerome A., Jr. ... Aberdeen
Eckrich, Jerome A., Sr. .. Aberdeen
Fahrenwald, Myron E. .... Aberdeen
Gerber, Bernard C. ..... Aberdeen
Harlow, Mark C. ..... Aberdeen
Hart, Harvey J. ..... Aberdeen
Heinemann, Phyllis E. .... Aberdeen
Holkesvick, R. E. ..... Aberdeen
Hovland, James I. ..... Aberdeen
Huber, Joel B. ..... Redfield
Janusz, Albin J. ..... Aberdeen
Kazi, K. Stephen ..... Aberdeen
Kom, Carlton J. ..... Aberdeen
Kosse, Karl H. ..... Aberdeen
Leon, Paul R. ..... Aberdeen
McFee, John L. ..... Ipswich
McGee, Robert C. ..... Aberdeen
McIntosh, George F. ..... Eureka
Myrmoe, Arlin M. ..... Aberdeen
*Norgello, Vikentijs .... Sioux Falls
Odland, Winston B. ..... Aberdeen
Ostrowski, Susan M. ..... Eureka
Patterson, David M. ..... Redfield
Pelton, Charles L. ..... Aberdeen
Redmond, Warren J. ..... Aberdeen
Rodine, John C. ..... Aberdeen
Sanders, Mary E. ..... Redfield
Scheffel, Alvin R. ..... Redfield
Seaman, David ..... Aberdeen
Shaw, Wayne ..... Aberdeen
Shinghal, Kumud K. ..... Aberdeen
Shinghal, Pramod ..... Aberdeen
Shousha, Alfred ..... Britton
Steele, Granville H. ..... Aberdeen
Stoppole, John A. ..... Aberdeen
Sweeny, William T. ..... Aberdeen
Tan, Raymundo T. ..... Aberdeen
Taylor, William R. ..... Aberdeen
Unite, Ismael H. ..... Aberdeen
Vogele, Alvin C. ..... Aberdeen
*Vogele, C. L. ..... Aberdeen
Welge, Barry G. ..... Aberdeen
Zvejnieks, Karlis ..... Aberdeen

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*Bartron, Harry J., Jr. ... Watertown
Brakss, Valdis ..... Watertown
Clark, Carroll J. ..... Watertown
Desai, Bhasker J. ..... Watertown
Engelhart, Kenneth E. ... Watertown
Fedt, Donald N. ..... Watertown
Frazer, Paul D. ..... Clear Lake
Gehring, S. ..... Watertown
Gerrish, Catherine ..... Watertown
Gerrish, Edwin ..... Watertown
Guddal, W. Nicol ..... Watertown
Hanson, Bernie H. P. ... Watertown
Hughes, Howard D. .... Clear Lake
*Huppler, E. G. ..... Minnesota
Lamb, Marlin ..... Watertown
Larson, James C. ..... Watertown
Larson, Paul M. ..... Watertown
Likness, Clark W. ..... Watertown
McClaflin, Richard R. ... Watertown
Meyer, Robert J. ..... Watertown
Michieli, Jose C. ..... Watertown
Nelson, Parry S. ..... Watertown
Nutt, Roger ..... Watertown
Peterson, Linda H. ..... Watertown
Piro, David F. ..... Watertown
Rittmann, John E. ..... Watertown
*Rousseau, Maurice C. .. Watertown
Ruggles, James G. ..... Watertown
Sperl, M. ..... Watertown
*Stoltz, C. Rodney ..... Sioux Falls
Stransky, John J. ..... Watertown
Thompson, M. George .. Watertown
Thompson, Marion C. ... Watertown
Tracy, Gerald E. ..... Watertown
Traub, Douglas ..... Watertown
Wegner, Edward ..... Watertown
Wrage, Theodore J., Jr. Watertown
Yemmanur, K. M. ..... Estelline
Yemmanur, S. D. ..... Estelline

MADISON-BROOKINGS
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Bandiera, Samuel J. ..... Brookings
*Friefeld, Saul ..... Minnesota
*Henry, Robert B. ..... Brookings
Holm, Richard P. ..... Brookings
Jacobs, Tad B. ..... Flandreau
*Kershner, Calvin N. .... Brookings
Lampert, Arthur A., Jr. ... Madison
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McHardy, Bryson R. .... Brookings
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*Peeke, Alonzo P. ..... Volga
Peik, Donald J. ..... Brookings
*Plowman, Elven T. ..... Brookings
Rietz, R. R. ..... Brookings
Roberts, Charles S., Jr. .. Brookings
Sample, Richard G. ..... Madison
Scheller, Donald L. ..... Arlington
Shaskey, Robert E. ..... Brookings
Stensrud, Homer J. ..... Madison
*Tank, Myron C. ..... Brookings
Tesch, Ronold R. ..... Brookings
Venugopal, Muthugounder Brookings
Wait, Curtis H. ..... Brookings
Wake, Richard A. ..... Brookings
Warren, Merrit G. ..... Brookings
Wilde, Kim L. ..... Madison

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Collins, E. Howard ..... Gettysburg
Cosand, Marion R. ..... Pierre
Davis, John B. ..... Pierre
Duggan, Robert ..... Pierre
*Fox, Stanley W. ..... Canada
Gregg, John ..... Sioux Falls
Hoffsten, Phillip E. ..... Pierre
Huber, Thomas J. ..... Pierre
Jahraus, R. Curtis ..... Pierre
Lindbloom, Buron O. ..... Pierre
Owens, Raymond J. ..... Pierre
Park, Dai H. ..... Pierre
Spears, Barbara K. ..... Pierre
Swanson, Charles L. ..... Pierre
Tieszen, Arden J. ..... Pierre
Warrender, C. L. ..... Pierre
Werthmann, Hubert E. ..... Pierre
Zakahi, Raymond J. ..... Pierre

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Belyea, Mark E. ..... Huron
Betts, L. S. ..... Huron
Buchanan, David J. ..... Huron
Cavanaugh, Dennis J. ..... Huron
Dean, Roscoe E. Wessington Springs
Dean, Thomas M. Wessington Springs
DeGeest, James H. ..... Miller
Gryte, Clifford F. ..... Huron
Hanson, William O. ..... Huron
Heilman, B. F. ..... Huron
*Hofer, Emil A. ..... Huron
Hohm, Paul H. ..... Huron
Hohm, Robert C. ..... Huron
Hohm, Theodore A. ..... Huron
Huet, William G. M. ..... Huron
Kapur, Hiroo R. ..... Huron
Kapur, Ravi ..... Huron
Karlen, Louis W. ..... DeSmet
Kim, Thomas H. ..... Huron
Kortum, Cynthia L. ..... Huron
Kurch, Julie Ann ..... Huron
Lardinois, Clifford C., Sr. ... Huron
Lenz, Bernard T. ..... Huron
Monfore, James E. ..... Miller
Nicholas, George A. ..... Huron
Saylor, Howard L., Jr. ..... Huron
Schneider, F. V. ..... Huron
Schroeder, Stephan D. ..... Miller
Sheekey, Owen J. ..... Huron

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Birkenkamp, Ray T. ..... Mitchell
Crandell, Michael P. ... Chamberlain
Dappen, Robert W. ..... Mitchell
Delaney, Robert J. ..... Mitchell
Delaney, William A., Jr. ... Mitchell
Dilger, Joseph T. ..... Mitchell
Flohr, Charles E. ..... Mitchell
Gaede, J. E. ..... Mitchell
Gere, Richard G. ..... Mitchell
Gillis, Floyd D., Jr. ..... Mitchell
Haley, Michael D. ..... Mitchell
Hays, Laura M. ..... Mitchell
Hermann, Harland T. ..... Mitchell
Heth, Samuel R. ..... Mitchell
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Howe, Jerome K. ..... Mitchell
Jones, John B. ..... Chamberlain
Judge, John O. ..... Mitchell
Kemp, James S. ..... Mitchell
Kramer, Charles G. Fort Thompson
*Lloyd, John H. ..... Arizona
Mabee, Judson O. ..... Mitchell
*Mabee, Oscar J. ..... Mitchell
Margallo, Lucio N., II. .... Mitchell
Moller, Christopher J. ..... Mitchell
Monson, Charles D. ..... Parkston
Mueller, Eric H. ..... Tripp
Olegario, Filemon E., Jr. .. Mitcheli
Phadke, Yeshwant G. .. Chamberlain
Porter, Maynard ..... Parkston
Ramos, Manuel D. ..... Scotland
Ramos, Manuel F. ..... Platte
Schabauer, Ernest A. ..... Mitchell
Silvagni, A. J. ..... Parkston
*Skogmo, Bernhoff R. .... Mitchell
*Tobin, Leonard W. ..... Mitchell
Van Ert, G. ..... Chamberlain
Visani, Sandro ..... Mitchell
*Vonburg, Vernon R. ..... Mitchell
Vose, James L. ..... Mitchell
Weatherill, Donald W. .... Mitchell
*Weber, Robert A. ..... Mitchell
Williams, H. Stephen ..... Mitchell

SIOUX FALLS
DISTRICT No. 7

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*Alcorn, Floyd A. ..... Sioux Falls
Alvine, Frank G. ..... Sioux Falls
Amundson, Loren H. ... Sioux Falls
Anderson, Courtney W. Sioux Falls
Anderson, Edward F. ... Sioux Falls
Anderson, Thorwald R. Sioux Falls
Anderson, Warren R. ... Sioux Falls
Angelos, Theodore A. ..... Canton
Arneson, Wallace A. ... Sioux Falls
Aspaas, Paul K., Jr. .... Sioux Falls
Aspaas, Paul K., Sr. ... Dell Rapids
Assam, Sam ..... Calif.
Augspurger, Ken D. .... Sioux Falls
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Barlow, John F. ..... Sioux Falls
Barnett, George L. ..... Sioux Falls
Belatti, Richard G. ..... Sioux Falls
Benson, Gail M. ..... Sioux Falls
Bess, Michael A. ..... Sioux Falls
Bhatti, Tajammul H. .... Sioux Falls
Billion, John J. ..... Sioux Falls
*Billion, Thomas J., Jr. Sioux Falls
Blake, Jerome M. ..... Sioux Falls
Boade, Werner A. ..... Sioux Falls
Brandenburg, Verdayne R. Sioux Falls
*Breit, Donald H. ..... Sioux Falls
Brown, Delbert L. ..... Sioux Falls
Bruins, George S. ..... Sioux Falls
Brzica, Stephen M. ..... Sioux Falls
Bucy, Christine F. ..... Sioux Falls
Burkhart, Thomas J. .... Sioux Falls
Burns, Edith A. ..... Sioux Falls
Burns, Howard W. ..... Sioux Falls
*Burns, Kendall R. ..... Sioux Falls
Busch, James ..... Sioux Falls
Carlisle, C. J. ..... Sioux Falls
*Carney, Myrtle ..... Texas
Carpenter, Paul L. ..... Sioux Falls
Carter, Guy A. ..... Sioux Falls
Chalmers, James H. .... Sioux Falls
Church, Bill ..... Sioux Falls
Cink, Thomas M. ..... Sioux Falls
Clark, Edward T. ..... Sioux Falls
*Cottam, G. I. W. ..... Sioux Falls
Cutshall, Vincent K. .... Sioux Falls
Daw, Edward F. ..... Sioux Falls
DeClark, Robert P. ..... Sioux Falls
Devick, John S. ..... Colton
*Dickinson, John H. ..... Canistota
Donahoe, John W. ..... Sioux Falls
Drymalski, Walter G. ... Sioux Falls
Dzintars, Valdis A. ..... Sioux Falls
Easton, Jessie K. M. .... Sioux Falls
*Eirinberg, Isadore D. .. Sioux Falls
Elkjer, Neil J. ..... Sioux Falls
Elson, David L. ..... Sioux Falls
English, Gilbert L. ..... Sioux Falls
Ensberg, Dorence L. .... Sioux Falls
Epp, Dennis L. ..... Freeman
Farrell, Harry W. ..... Sioux Falls
Felker, James R. ..... Sioux Falls
Fenton, Lawrence J. .... Sioux Falls
Ferrell, Michael R. ..... Sioux Falls
Finney, Lawrence W. ... Sioux Falls
*Fisk, Robert G. ..... Flandreau
Flora, George C. ..... Sioux Falls
Foss, J. Frank ..... Sioux Falls
Freeman, Jerome W. .... Sioux Falls
Friess, Richard W. ..... Sioux Falls
Frost, Donald M. ..... Sioux Falls
Fryda, Wm. C. ..... Sioux Falls
Fuller, William C. ..... Sioux Falls
Gaeckle, Thomas ..... Sioux Falls
Giebink, Robert R. ..... Sioux Falls
Graham, Donald B. ..... Sioux Falls
Grau, Thomas J. ..... Sioux Falls
Green, Marc ..... Sioux Falls
Greenfield, Duane L. ... Sioux Falls
Groote, Curtis A. ..... Sioux Falls
Gross, H. Phil ..... Sioux Falls
*Grove, M. Stuart ..... Sioux Falls
Gunnarson, Richard E. .. Sioux Falls
Gutch, Charley F. ..... Sioux Falls
Gutnik, Leonard M. .... Sioux Falls
Hagen, Jeffrey B. ..... Sioux Falls
Harms, R. W. ..... Sioux Falls
Harris, F. L. ..... Sioux Falls
Harris, Mary ..... Sioux Falls
Hartmann, Alfred E. .... Sioux Falls
Hartzell, Allan J. ..... Sioux Falls
Held, Gordon R. ..... Sioux Falls
Henrickson, Lynn A. ... Sioux Falls
Henrickson, Robert G. .. Sioux Falls
Hohm, Byron T. ..... Sioux Falls
Horner, William ..... Sioux Falls
Horning, J. R. ..... Sioux Falls
Hosen, Richard S. ..... Sioux Falls
Hoskins, John H. ..... Sioux Falls
Hoversten, David L. .... Sioux Falls
Hoxtell, Eugene O. ..... Sioux Falls
Humphreys, Donald W. Sioux Falls

Hurley, Brian ..... Sioux Falls
Hurley, Timothy ..... Sioux Falls
Hussain, Rif'at ..... Sioux Falls
Hyland, Lowell J. ..... Sioux Falls
Ingvoldstad, James P. ... Sioux Falls
Janis, John B. ..... Sioux Falls
Jaqua, Richard A. ..... Sioux Falls
Javurek, Anthony J. .... Sioux Falls
Johnson, Beth L. ..... Sioux Falls
Johnson, Dennis L. ..... Sioux Falls
Johnson, Robert C. ..... Sioux Falls
Jones, Warren L. ..... Sioux Falls
Justice, Michael W. .... Dell Rapids
Kalda, Ellison F., II .... Sioux Falls
Kangley, Daniel J. ..... Sioux Falls
Kaufman, Irvin I. ..... Freeman
Kemp, Earl D. ..... Sioux Falls
Kennelly, Daniel J. ..... Sioux Falls
*King, Lyndon M., Jr. .. Sioux Falls
Kittelson, H. Otis ..... Sioux Falls
Knowles, Roy C. ..... Sioux Falls
Knudson, Donald H. .... Sioux Falls
Knutson, Dennis D. .... Sioux Falls
*Kohlmeyer, Frederick C. Sioux Falls
Koob, K. Gene ..... Sioux Falls
Lakstigala, Peters E. .... Sioux Falls
Lang, Durward M. ..... Omaha, NE
Langdon, John G. ..... Sioux Falls
Lankhorst, Barry J. ..... Sioux Falls
Larke, Daryl S. ..... Mich.
Larson, Leland J. ..... Sioux Falls
*Leander, Richard B. ... Sioux Falls
Lee, Si Graph ..... Sioux Falls
Looby, Thomas L. ..... Sioux Falls
Loos, Gerald D. ..... Sioux Falls
Lovrien, Fred C. ..... Sioux Falls
Lowery, Gayla S. ..... Sioux Falls
Luechtefeld, Nancy ..... Sioux Falls
Mabee, Lee ..... Sioux Falls
Madison, Dean L. ..... Sioux Falls
Magnuson, Gregory L. .. Sioux Falls
*Maresh, Everett R. .... Sioux Falls
Mark, Curtis L. ..... Viborg
Marschke, Robert F., Jr. Sioux Falls
Masterson, Thomas E. .. Sioux Falls
Matustik, M. C. ..... Sioux Falls
McGrann, James R. .... Sioux Falls
*McGreevy, John V. ... Sioux Falls
McGreevy, Patrick S. ... Sioux Falls
McKercher, Scott ..... Sioux Falls
McMillin, J. Michael ... Sioux Falls
Mohler, Charles Wm. ... Sioux Falls
Morris, Alan D. ..... Sioux Falls
Munson, David P. ..... Sioux Falls
Mutch, Milton G. ..... Sioux Falls
Naughton, Gregory ..... Sioux Falls
Neidich, Gary ..... Sioux Falls
Nelimark, Robert A. .... Sioux Falls
Nelson, Earl G. ..... Viborg
Nelson, Richard A. ..... Sioux Falls
Nelson, Robert E. ..... Sioux Falls
Nice, Richard F. ..... Sioux Falls
Nielsen, James L. ..... Dell Rapids
Nordstrom, Donald G. .. Sioux Falls
Oakland, James A. ..... Sioux Falls
O'Brien, Charles P. .... Sioux Falls
O'Brien, Peter J. ..... Sioux Falls
Ochsner, John A. ..... Sioux Falls
Ofstein, Lewis C. ..... Sioux Falls
*Ogborn, Robert J. ..... Sioux Falls
Ohrt, David W. ..... Sioux Falls
Olson, Michael L. ..... Sioux Falls
*Opheim, Warren L. .... Sioux Falls
Opheim, Warren O. V. .. Sioux Falls
Orr, Russell T. ..... Sioux Falls
Ortmeier, Denny G. .... Sioux Falls
Owens, Leycester, Jr. ... Sioux Falls
Parry, Rodney R. ..... Sioux Falls
*Pasek, Edward A. ..... Sioux Falls
Payne, Harlan A. ..... Sioux Falls
Pekas, Michael W. ..... Sioux Falls
Petereit, Martin F. ..... Sioux Falls
Peters, Edward H. ..... Sioux Falls
Peters, Patricia ..... Sioux Falls
Peterson, Karl G. ..... Sioux Falls
Petres, Anthony ..... Salem
Pitt-Hart, Barry T. ..... Sioux Falls
Plummer, Richard ..... Sioux Falls
Putnam, Wesley D. ..... Sioux Falls
Quale, James L. ..... Sioux Falls
Quinn, Robert H. ..... Sioux Falls
Randall, Bradley B. .... Sioux Falls
Raszkowski, Robert R. .. Sioux Falls
Read, Ralph L. ..... Sioux Falls
Regier, Eugene R. ..... Canton
Reynolds, James R. .... Sioux Falls
Richards, George A. .... Sioux Falls
Rodman, Peter K. ..... Sioux Falls
Rolfsmeyer, Eric ..... Sioux Falls
Rossing, David R. ..... Sioux Falls
Rossing, William O. .... Sioux Falls
Rost, Michael C. ..... Sioux Falls
Ryan, James E. ..... Sioux Falls
Rydberg, Mitchell L. ... Dell Rapids
Salem, Anthony G. ..... Sioux Falls
Sall, John C. ..... Sioux Falls
Salmela, Steven R. ..... Sioux Falls
Sanchez, Gonzalo M. ... Sioux Falls
Sanderson, Everett W. .. Sioux Falls
Schellpfeffer, Donald ... Sioux Falls
Schultz, Richard D. .... Sioux Falls
Schultz, Thomas ..... Sioux Falls
Simmons, Jerry L. ..... Sioux Falls
Sittner, Larry ..... Sioux Falls
Slattery, Mary T. ..... Sioux Falls
*Smith, George ..... Missouri
Solberg, Lloyd E. ..... Sioux Falls
Soye, Andrew I. ..... Sioux Falls
*Stahmann, Fred S. .... Sioux Falls
Stassen, Michael D. .... Sioux Falls
Steidl, Lester ..... Sioux Falls
*Steiner, Peter K. ..... California
Stensland, Vernon H. ... Sioux Falls
*Stern, Charles A. ..... California
Stevens, Dennis C. ..... Sioux Falls
Stoltz, C. Roger ..... Sioux Falls
Talley, Robert C. ..... Sioux Falls
Tam, Guy E. ..... Sioux Falls
Tieszen, Jerel E. ..... Sioux Falls
Tobin, Michael D. ..... Sioux Falls
Tschetter, Loren K. ..... Sioux Falls
Tschetter, Richard T. ... Sioux Falls
VanDemark, R. E., Sr. . Sioux Falls
VanDemark, Robert E., Jr. Sioux Falls
VanderWoude, Larry B. Sioux Falls
Villa, Jose P. ..... Freeman
Vogt, H. Bruce ..... Sioux Falls
Volin, Verlynne V. ..... Sioux Falls
Wagner, Loyd R. ..... Sioux Falls
Waltner, Lonnie L. .... Bridgewater
Walton, Jerry L. ..... Sioux Falls
Watson, William V. .... Sioux Falls
Wegner, Karl H. ..... Sioux Falls
White, Thomas C. ..... Sioux Falls
Wierda, Daryl R. ..... Sioux Falls
Williams, Buck J. ..... Sioux Falls
Wilson, Thomas M. .... Sioux Falls
Wirtz, Patricia S. ..... Sioux Falls
Wyatt, George W. ..... Sioux Falls
Wyatt, Ronald O. ..... Sioux Falls
Zawada, E. T. ..... Sioux Falls

YANKTON
DISTRICT No. 8

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Adams, Curtis M. ..... Yankton
Bean, David W. ..... Yankton
Bentz, J. W. ..... Platte
Brookman, Bruce T. ..... Wagner
Buchhammer, Carlos R. Vermillion
Dendinger, William J. ... Vermillion
Fletcher, Harold J. ..... Vermillion
Flom, Jon O. ..... Yankton
Foley, Robert J. ..... Tyndall
Gunderson, Dale E. ..... Yankton
Halverson, Kenneth ..... Yankton
Heinrichs, Eberhard H. .. Vermillion
Holzwarth, David R. ..... Yankton
Honke, Richard W., II. .... Wagner
Hubner, Jay W. ..... Yankton
*Hubner, Roland F. ..... Yankton
Isburg, Carroll D. ..... Yankton
Jacobsen, John J. ..... Yankton
Jameson, G. Malcolm ..... Yankton
Johnson, Beth ..... Vermillion
Johnson, Thomas C. ..... Yankton
Johnson, Virginia P. .... Vermillion
Kalda, Ellison F. ..... Platte
Lyso, Melford B. ..... Yankton
*McVay, Chester B. ..... Yankton
McVay, Michael R. ..... Yankton
Messner, Frank D. ..... Yankton
Meyer, Larry A. ..... Yankton
Neumayr, Robert J. ..... Yankton
Olson, Thomas H. ..... Vermillion
Pascale, Carl C. ..... Centerville
Pesce, Ulises ..... Yankton
Petersen, Loren P. ..... Yankton
Porter, Richard I. ..... Yankton
Price, Ronald D. ..... Armour
Provow, Susan R. ..... Vermillion
Quick, William W. ..... Yankton
Radack, Morris L. ..... Yankton
Ranney, Brooks ..... Yankton
Reaney, Duane B. ..... Yankton
Reding, Arthur P. ..... Marion
Rhoades, Marques E. ..... Yankton
*Riesberg, Elsa ..... Aberdeen
Saloum, Herbert T. ..... Tyndall
Saoi, Nicasio B. ..... Yankton
Sattler, Theodore H. ..... Yankton
*Sebring, Floyd U. ..... Minn.
Smith, David A. ..... Yankton
Stanage, Willis F. ..... Pierre
Steele, James P. ..... Yankton
Sternquist, John C. ..... Yankton
Stevens, Julie C. ..... Yankton
Thompson, Robert F. ..... Yankton
Tidd, John T. ..... Yankton
Tuan, Chung H. ..... Yankton
Turner, Charles R. ..... Vermillion
Willcockson, John R. ..... Yankton
Willcockson, Thomas H. ... Yankton

BLACK HILLS
DISTRICT No. 9

Pres., Richard Renka, M.D. — Vice Pres., Robert Allen, M.D. — Sec., N. R. Whitney, M.D.

Ahrlin, H. Lee, Jr. ..... Rapid City
Ahrlin, Hollis L. ..... Rapid City
Allen, Bruce H. ..... Rapid City
Allen, Robert G. ..... Rapid City
Anderson, A. Byford .... Deadwood
Arnold, George H. ..... Rapid City
Authier, Noe ..... Rapid City
*Bailey, John D. ..... Rapid City
Bareis, Reuben J. ..... Rapid City
Bauman, Randell E. .... Rapid City
Bedingfield, John R. .... Rapid City
Behrens, Clayton L. .... Rapid City
Bergeron, Dale A. ..... Rapid City
Berkebile, Dale E. ..... Rapid City
Bloemendaal, Robert D. Rapid City
*Borgmeyer, Henry J. ... Rapid City
Boyce, Raymond A. .... Rapid City
Boyer, David W. ..... Rapid City
Branch, Robert F. ..... Rapid City
*Bray, Robert B. ..... Rapid City
Brown, Michael ..... Spearfish
Burnett, Raymond G. ... Rapid City
Butz, Gerald W. ..... Rapid City
Calhoon, Stephen L. .... Rapid City
Carlson, Gary L. ..... Rapid City
Carson, Layne E. .. Little Rock, AR
*Clark, Bernard S. ..... Spearfish
*Cline, James A. Rocky Mount, NC
Cornford, Raymond C. .. Rapid City
Dewald, Allan L. ..... Rapid City
Dickson, Daryl S. ..... Hot Springs
Drummond, Ronald G. .. Rapid City
Dzintars, Paul F. ..... Rapid City
Ebbert, Larry P. ..... Rapid City
Elk, Sidney A ..... Rapid City
Elston, John T. ..... Rapid City
Engelbrecht, J. A. ..... Rapid City
Ferrell, Robert L. ..... Rapid City
Finley, Richard C. ..... Rapid City
Freimark, Lyle G. ..... Rapid City
Fromm, Harold E. ..... Rapid City
Frost, Harold L. ..... Rapid City
*Gilbert, Freeman J. .. Belle Fourche
Gill, Timothy J. ..... Rapid City
Golliher, Warren N. ..... Spearfish
Goodhope, Robert C. ... Fort Meade
Gwinn, Charles B. ..... Rapid City
Haas, Stephen N. ..... Rapid City
Hafner, Daniel J. ..... Rapid City
Hamm, Joseph N. ..... Rapid City
Hansen, Craig K. ..... Rapid City
Hare, Helen Jane ..... Rapid City
Harris, Russell H. ..... Rapid City
Haugan, Haakon O. ..... Rapid City
Haydon, J. R. ..... Nebraska
Hayes, Robert H. ..... Wall
Heidepriem, Glen ..... Rapid City
Hercules, Costas ..... Rapid City
Herlihy, J. J. ..... Rapid City
Hewitt, John M. ..... Rapid City
Howard, William J. ..... Rapid City
Jackson, James W. ..... Rapid City
Jacobson, Theodore R. Hot Springs
James, Edward H. ..... Rapid City
Jenter, George W. ..... Sturgis
Jentes, Paul K. ..... Fort Meade
Jerde, O. Myron ..... Rapid City
Johnson, Robert K. ..... Rapid City
Jones, William E. ..... Sturgis
Kelley, Donald H. ..... Rapid City
Kelts, K. Alan ..... Rapid City
Klar, Werner ..... Fort Meade
Knecht, John F. ..... Martin
*Koren, Paul H. ..... Rapid City
Kovarik, Joseph A. ..... Rapid City
Kovarik, Richard A. .... Rapid City
Kovarik, Wenzel J. ..... Rapid City
Krafka, Thomas L. ..... Rapid City
Kullbom, James B. ..... Rapid City
Kunz, James A. ..... Rapid City
Kwan, Francis P. ..... Rapid City
*Lampert, Arthur A. .... Rapid City
Lauer, David A. ..... Sturgis
Liedtke, Curtis J. ..... Sturgis
Loos, Charles M. ..... Rapid City
Lopez, Alberto S. ..... Hot Springs
Mangulis, George J. ..... Philip
Manzo-Chua, D. ..... Hot Springs
Markusfeld, Jack M. .... Rapid City
Marshall, John ..... Rapid City
Massa, Lawrence L. ..... Sturgis
Mattson, William J. ..... Rapid City
McGuigan, Patrick M. ... Rapid City
Melvin, Gary ..... Fort Meade
*Merryman, Murlin P. .. Rapid City
Millea, Roger P. ..... Rapid City
Mortimer, Sam L. ..... Rapid City
Munson, H. Benjamin ... Rapid City
Neu, Norman D. ..... Rapid City
Nord, Allen E. ..... Rapid City
Norris, M. G. ..... Hot Springs
Oliver, D. E. ..... Rapid City
Owen, Gordon S. ..... Rapid City
Palmerton, Ernest S. .... Rapid City
*Perry, Wm. J. ..... Rapid City
Pieczka, Jan K. ..... Hot Springs
Pucelik, J. A. ..... Deadwood
Reagan, James ..... Sturgis
Reinoehl, Warren L. ..... Custer
Renka, Richard P. ..... Rapid City
Ridenour, Chester D. ..... Sturgis
Rosario, Elmo J. ..... Rapid City
Rud, James A. ..... Rapid City
Ruud, Edward T. ..... Hot Springs
Sandvik, David E. ..... Rapid City
Sanmartin, Jorge E. ..... Rapid City
*Saxton, A. J. ..... New Mexico
Sejvar, Joseph P. ..... Rapid City
Shining, H. Streeter ..... Rapid City
Silenas, Raza ..... Rapid City
Slingsby, J. Geoffrey .... Rapid City
Slingsby, John B. ..... Rapid City
Strand, Ray D. ..... Rapid City
Sutliff, Willis C. ..... Rapid City
Swisher, Lowell P. ..... Kadoka
Theissen, Hubert H. .... Rapid City
Trinidad, Reuben B. ..... Lead
Tschetter, William R. ... Rapid City
VanEtten, Donald D. .... Rapid City
Vogele, Kenneth A. ..... Rapid City
Weitzenkamp, Larry .... Rapid City
Welsh, Gary L. ..... Rapid City
Westaby, Robert S. .... Hot Springs
Whitney, Nathaniel R. ... Rapid City
Wicks, Dennis R. ..... Custer
Wieland, Clark ..... Fort Meade
*Williams, F. R. ..... Arizona
Williams, Robert ..... Rapid City
Wingert, Robert I. ..... Rapid City
Wood, George F., Jr. ... Rapid City
Wright, Paul L. ..... Rapid City
Wudel, R. L. ..... Spearfish
Yackley, James V. ..... Rapid City
Yamada, Andrew R. .... Rapid City
Zanka, J. A. ..... Rapid City
Zielike, Carol M. ..... Rapid City

ROSEBUD
DISTRICT No. 10

Pres., Gregg Tobin, M.D. — Vice. Pres., Tony Berg, M.D. — Sec., R. G. Nemer, M.D.

Berg, Tony L. ..... Winner
Hogrefe, Louis H. ..... Gregory
Malm, John A. ..... Gregory
Marquardt, Clark ..... Rosebud
Nemer, Raymond G. ..... Gregory
Stiehl, Robert L. ..... Winner
Sweet, Edwin P. ..... Burke
Tobin, Gregg ..... Winner

NORTHWEST
DISTRICT No. 11

Pres., Ben Henderson, D.O. — Vice Pres., Jeffrey Peterson, M.D. — Sec., L. M. Linde, M.D.

Collins, James D. ..... Mobridge
Henderson, Ben J. ..... Mobridge
Johnson, Charles A. ..... Lemmon
Linde, Leonard M. ..... Mobridge
*Nolan, Bernard P. ..... Mobridge
Peterson, Jeffrey L. ..... Mobridge
*Spiry, Arthur W. ..... Mobridge
Wunder, James F. ..... Mobridge
Yecha, David J. ..... Gettysburg

WHETSTONE VALLEY
DISTRICT No. 12

Pres. V. Vanadurongvan, M.D. Sec., K. Vanadurongvan, M.D.

Bell, Eldon E. ..... Webster
*Gregory, David A. ..... Montana
Janavs, Visvaldis ..... Milbank
Johnson, Edward A. ..... Milbank
Kass, Joseph ..... Rosholt
Kosa, A. ..... Webster
Nelson, Lawrence F. ..... Webster
Oey, David L. ..... Sisseton
Staub, David W. ..... Sisseton
Vanadurongvan, K. ..... Milbank
Vanadurongvan, V. ..... Milbank
Vogelgesang, Lloyd C. .... Webster

* Indicates Honorary Member

South Dakota State Medical Association Roster — 1984 Membership — Alphabetical Listing

Aanning, Harold L. ..... Yankton
Abu-Ghazaleh, Samir Z. Sioux Falls
Adams, Curtis M. ..... Yankton
Adams, Harold P. ..... Huron
Ahrlin, H. Lee, Jr. ..... Rapid City
Ahrlin, Hollis L. ..... Rapid City
Albano, Paterno C. ..... Aberdeen
*Alcorn, Floyd ..... Sioux Falls
Alexander, James R. ..... Aberdeen
Allen, Bruce H. ..... Rapid City
Allen, Robert G. ..... Rapid City
Allen, Stanley W., Jr. ... Watertown
Altman, Stanley B. ..... Aberdeen
Alvine, Frank G. ..... Sioux Falls
Amundson, Loren H. ... Sioux Falls
Andersen, Calvin F. ..... Aberdeen
Anderson, A. Byford .... Deadwood
Anderson, Courtney W. Sioux Falls
Anderson, Edward F. ... Sioux Falls
Anderson, Esther E. ..... Aberdeen
Anderson, James A. ..... Madison
Anderson, Thorwald R. Sioux Falls
Anderson, Warren R. ... Sioux Falls
Angelos, Theodore A. ..... Canton
Appelwick, James E. ..... Madison
Argabrite, John W. ..... Watertown
Arneson, Wallace A. ... Sioux Falls
Arnold, George H. ..... Rapid City
*Askwig, Leroy C. ..... Pierre
Aspaas, Paul K., Jr. .... Sioux Falls
Aspaas, Pauk K., Sr. ... Dell Rapids
Assam, Sam ..... Calif.
Augspurger, Ken D. .... Sioux Falls
Authier, Noe ..... Rapid City

Baas, Walter P. ..... Mitchell
Bachmayer, Jay D. ..... Aberdeen
*Bailey, John D. ..... Rapid City
Baltodano, N. ..... Lake Preston
Bandiera, Samuel J. ..... Brookings
Bareis, Reuben J. ..... Rapid City
Barker, John D., Jr. .... Sioux Falls
Barlow, John F. ..... Sioux Falls
Barnett, George L. ..... Sioux Falls
Bartholomew, Kenneth A. Faulkton
Bartron, G. Robert ..... Watertown
*Bartron, Harry J., Jr. ... Watertown
Bauman, Randell E. .... Rapid City
Bean, David W. ..... Yankton
Bedingfield, John R. .... Rapid City
Behrens, Clayton L. .... Rapid City
Belatti, Richard G. ..... Sioux Falls
Bell, Eldon E. ..... Webster
Bell, G. Robert ..... DeSmet
Belyea, Mark E. ..... Huron
Benson, Gail M. ..... Sioux Falls
Bentz, J. W. ..... Platte
Berg, Sterling ..... Redfield
Berg, Tony L. ..... Winner
Bergeron, Dale A. ..... Rapid City
Berkebile, Dale E. ..... Rapid City
Berry, Jack T. ..... Mitchell
Bess, Michael A. ..... Sioux Falls
Betts, L. S. ..... Huron
Bhat, Dileep S. ..... Mitchell
Bhatti, Tajammul H. .... Sioux Falls
Billion, John J. ..... Sioux Falls
*Billion, Thomas J. .... Sioux Falls
Binder, Clifford F. .... Chamberlain
Birkenkamp, Ray T. ..... Mitchell
Blake, Jerome M. ..... Sioux Falls
*Bloemendaal, Gerrit J. .... Ipswich
Bloemendaal, Robert D. Rapid City
Boade, Werner A. ..... Sioux Falls
*Borgmeyer, Henry J. ... Rapid City
Boyce, Raymond A. .... Rapid City
Boyer, David W. ..... Rapid City
Brakss, Valdis ..... Watertown
Branch, Robert F. ..... Rapid City
Brandenburg, Verdayne R. Sioux Falls
*Bray, Robert ..... Rapid City
*Breit, Donald H. ..... Sioux Falls
Brevik, Alan K. ..... Redfield
Broadhurst, Kennon E. ... Aberdeen
Brookman, Bruce T. ..... Wagner
Brown, Bradley A. ..... Watertown
Brown, Delbert L. ..... Sioux Falls
Brown, Michael ..... Spearfish
Brown, Robert H. ..... Canada
Bruins, George S. ..... Sioux Falls
Brzica, Stephen M. ..... Sioux Falls
Buchanan, David J. ..... Huron
Buckhammer, Carlos R. Vermillion
Bucy, Christine F. ..... Sioux Falls
Burkhart, Thomas J. .... Sioux Falls
Burnett, Raymond G. ... Rapid City
Burns, Edith A. ..... Sioux Falls
Burns, Howard W. ..... Sioux Falls
*Burns, Kendall R. ..... Sioux Falls
Busch, James ..... Sioux Falls
Butz, Gerald W. ..... Rapid City

Calhoon, Stephen L. .... Rapid City
Carlisle, Christopher ..... Salem
Carlson, Gary L. ..... Rapid City
*Carney, Myrtle ... Fort Worth, TX
Carpenter, Paul L. ..... Sioux Falls
Carson, Layne E. .. Little Rock, AR
Carter, Guy A. ..... Sioux Falls
Carter, Peter B. ..... Aberdeen
Cavanaugh, Dennis J. ..... Huron
Chalmers, James H. .... Sioux Falls
Chang, Joe P. ..... Aberdeen
Chavier, Juan R. ..... Aberdeen
Chen, Cheng-Fu ..... Aberdeen
Christopher, John R. ..... Aberdeen
Church, Bill ..... Sioux Falls
Cink, Thomas M. ..... Sioux Falls
*Clark, Bernard S. ..... Spearfish
Clark, Carroll J. ..... Watertown
Clark, Edward T. ..... Sioux Falls
*Cine, James A. Rocky Mount, NC
Colberg, Douglas ..... Aberdeen
Collins, E. Howard ..... Gettysburg
Collins, James D. ..... Mobridge
Cornford, Raymond C. .. Rapid City
Cosand, Marion R. ..... Pierre
*Cottam, G. I. W. ..... Sioux Falls
Crandell, Michael P. ... Chamberlain
Cutshall, Vincent K. .... Sioux Falls

Dappen, Robert W. ..... Mitchell
Davis, John B. ..... Pierre
Daw, Edward F. ..... Sioux Falls
Dean, Roscoe E. Wessington Springs
Dean, Thomas M. Wessington Springs
DeClark, Robert P. ..... Sioux Falls
DeGeest, James H. ..... Miller
Delaney, Robert J. ..... Mitchell
Delaney, William A., Jr. ... Mitchell
Dendinger, William J. ... Vermillion
Desai, Bhasker J. ..... Watertown
Devick, John S. ..... Colton
Dewald, Allan L. ..... Rapid City
*Dickinson, John ..... Canistota
Dickson, Daryl S. ..... Hot Springs
Dilger, Joseph T. ..... Mitchell
Donahoe, John W. ..... Sioux Falls
Drummond, Ronald G. .. Rapid City
Drymalski, Walter G. ... Sioux Falls
D'Souza, Edward P. ..... Aberdeen
Duggan, Robert ..... Pierre
Dzintars, Paul F. ..... Rapid City
Dzintars, Valdis A. ..... Sioux Falls

Easton, Jessie K. M. .... Sioux Falls
Ebbert, Larry P. ..... Rapid City
Eckrich, Jerome A., Jr. ... Aberdeen
Eckrich, Jerome A., Sr. .. Aberdeen
*Eirinberg, Isadore D. .. Sioux Falls
Elk, Sidney A. ..... Rapid City
Elkjer, Neil J. ..... Sioux Falls
Elson, David L. ..... Sioux Falls
Elston, John T. ..... Rapid City
Engelbrecht, J. A. ..... Rapid City

Engelhart, Kenneth E. ... Watertown
English, Gilbert. ..... Sioux Falls
Ensberg, Dorence L. .... Sioux Falls
Epp, Dennis L. ..... Freeman

Fahrenwald, Myron E. .... Aberdeen
Farrell, Harry W. ..... Sioux Falls
Fedt, Donald N. ..... Watertown
Felker, James R. ..... Sioux Falls
Fenton, Lawrence J. .... Sioux Falls
Ferrell, Michael R. ..... Sioux Falls
Ferrell, Robert L. ..... Rapid City
Finley, Richard C. ..... Rapid City
Finney, Lawrence W. ... Sioux Falls
*Fisk, Robert G. ..... Flandreau
Fletcher, Harold J. ..... Vermillion
Flohr, Charles E. ..... Mitchell
Flom, Jon O. ..... Yankton
Flora, George C. ..... Sioux Falls
Foley, Robert J. ..... Tyndall
Foss, J. Frank ..... Sioux Falls
*Fox, Stanley W. ..... Canada
Frazer, Paul D. ..... Clear Lake
Freeman, Jerome W. .... Sioux Falls
Freimark, Lyle G. ..... Rapid City
*Friefeld, Saul ... Minneapolis, MN
Friess, Richard W. ..... Sioux Falls
Fromm, Harold E. ..... Rapid City
Frost, Donald M. ..... Sioux Falls
Frost, Harold L. ..... Rapid City
Fryda, Wm. C. ..... Sioux Falls
Fuller, William C. ..... Sioux Falls

Gaeckle, Thomas ..... Sioux Falls
Gaede, James ..... Mitchell
Gehring, Stephen H. .... Watertown
Gerber, Bernard C. ..... Aberdeen
Gere, Richard G. ..... Mitchell
Gerrish, Catherine ..... Watertown
Gerrish, Edwin ..... Watertown
Giebink, Robert R. ..... Sioux Falls
*Gilbert, Freeman J. .. Belle Fourche
Gill, Timothy J. ..... Rapid City
Gillis, Floyd D., Jr. ..... Mitchell
Golliher, Warren N. ..... Spearfish
Goodhope, Robert C. ... Fort Meade
Graham, Donald B. ..... Sioux Falls
Grau, Thomas J. ..... Sioux Falls
Green, Marc ..... Sioux Falls
Greenfield, Duane L. ... Sioux Falls
Gregg, John B. ..... Sioux Falls
*Gregory, David A. .. Glasgow, MT
Groote, Curtis A. ..... Sioux Falls
Gross, H. Phil ..... Sioux Falls
*Grove, M. Stuart ..... Sioux Falls
Gryte, Clifford F. ..... Huron
Guddal, W. Nicol ..... Watertown
Gunderson, Dale E. ..... Yankton
Gunnarson, Richard E. .. Sioux Falls
Gutch, Charley F. ..... Sioux Falls
Gutnik, Leonard M. .... Sioux Falls
Gwinn, Charles B. ..... Rapid City

Haas, Stephen N. ..... Rapid City
Hafner, Daniel J. ..... Rapid City
Hagen, Jeffrey B. ..... Sioux Falls
Haley, Michael D. ..... Mitchell
Halverson, Kenneth ..... Yankton
Hamm, Joseph N. ..... Rapid City
Hansen, Craig K. ..... Rapid City
Hanson, Bernie H. P. ... Watertown
Hanson, William O. ..... Huron
Hare, Helen Jane ..... Rapid City
Harlow, Mark C. ..... Aberdeen
Harms, R. W. ..... Sioux Falls
Harris, F. L. ..... Sioux Falls
Harris, Mary ..... Sioux Falls
Harris, Russell H. ..... Rapid City
Hart, Harvey J. ..... Aberdeen
Hartmann, Alfred E. .... Sioux Falls
Hartzell, Allan J. ..... Sioux Falls
Haugan, Haakon O. ..... Rapid City
Haydon, J. R. ..... Nebraska
Hayes, Robert H. ..... Wall
Hays, Laura M. ..... Mitchell
Heidepriem, Glen ..... Rapid City
Heilman, B. F. ..... Huron
Heinemann, Phyllis E. .... Aberdeen
Heinrichs, Eberhard H. .. Vermillion
Held, Gordon R. ..... Sioux Falls
Henderson, Ben J. ..... Mobridge
Henrickson, Lynn A. ... Sioux Falls
Henrickson, Robert G. .. Sioux Falls
*Henry, Robert B. ..... Brookings
Henry, Thomas E. ..... Tucson, AZ
Hercules, Costas ..... Rapid City
Herlihy, J. J. ..... Rapid City
Hermann, Harlan T. ..... Mitchell
Heth, Samuel R. ..... Mitchell
Hewitt, John M. ..... Rapid City
Hockett, Richard D. ..... Mitchell
*Hofer, Emil A. ..... Huron
Hoffsten, Phillip E. ..... Pierre
Hogrefe, Louis H. ..... Gregory
Hohm, Byron T. ..... Sioux Falls
Hohm, Paul H. ..... Huron
Hohm, Robert C. ..... Huron
Hohm, Theodore A. ..... Huron
Holkesvick, R. E. ..... Aberdeen
Holland, Lambert W. .. Chamberlain
Holm, Richard P. ..... Brookings
Holzwarth, David R. ..... Yankton
Honke, Richard W., II ..... Wagner
Horner, William ..... Sioux Falls
Horning, James R. ..... Sioux Falls
Hosen, Richard S. ..... Sioux Falls
Hoskins, John H. ..... Sioux Falls
Hoversten, David L. .... Sioux Falls
Hovland, James I. ..... Aberdeen
Howard, William J. ..... Rapid City
Howe, Jerome K. ..... Mitchell
Hoxtell, Eugene O. ..... Sioux Falls
Huber, Joel B. ..... Redfield
Huber, Thomas J. ..... Pierre
Hubner, Jay W. ..... Yankton
*Hubner, Roland F. ..... Yankton
Huet, William G. M. ..... Huron
Hughes, Howard D. .... Clear Lake
Humphreys, Donald W. Sioux Falls
*Huppler, Edward G. St. Paul, MN
Hurley, Brian ..... Sioux Falls
Hurley, Timothy ..... Sioux Falls
Hussain, Rif'at ..... Sioux Falls
Hyland, Lowell J. ..... Sioux Falls

Ingvoldstad, James P. ... Sioux Falls
Isburg, Carroll D. ..... Yankton

Jackson, James W. ..... Rapid City
Jacobs, Tad B. ..... Flandreau
Jacobsen, John J. ..... Yankton
Jacobson, Theodore R. Hot Springs
Jahraus, R. Curtis ..... Pierre
James, Edward H. ..... Rapid City
Jameson, G. Malcolm ..... Yankton
Janavs, Visvaldis ..... Milbank
Janis, John B. ..... Sioux Falls
Janusz, Albin J. ..... Aberdeen
Jaqua, Richard A. ..... Sioux Falls
Javurek, Anthony J. .... Sioux Falls
Jenter, George W. ..... Sturgis
Jentes, Paul K. ..... Fort Meade
Jerde, O. Myron ..... Rapid City
Johnson, Beth L. ..... Sioux Falls
Johnson, Charles A. ..... Lemmon
Johnson, Dennis L. ..... Sioux Falls
Johnson, Edward A. ..... Milbank
Johnson, Robert C. ..... Sioux Falls
Johnson, Robert K. ..... Rapid City
Johnson, Thomas C. ..... Yankton
Johnson, Virginia P. .... Vermillion
Jones, John B. ..... Chamberlain
Jones, Warren L. ..... Sioux Falls
Jones, William E. ..... Sturgis
Judge, John O. ..... Mitchell
Justice, Michael W. .... Dell Rapids

Kalda, Ellison F. ..... Platte
Kalda, Ellison F., II .... Sioux Falls
Kangley, Daniel J. ..... Sioux Falls
Kapur, Hiroo R. ..... Huron
Kapur, Ravi ..... Huron
Karlen, Louis W. ..... DeSmet
Kass, Joseph ..... Rosholt
Kaufman, Irvin I. ..... Freeman
Kazi, K. Stephen ..... Aberdeen
Kelley, Donald H. ..... Rapid City
Kelts, K. Alan ..... Rapid City
Kemp, Earl D. ..... Sioux Falls
Kemp, James S. ..... Mitchell
Kennelly, Daniel J. ..... Sioux Falls
*Kershner, Calvin M. .... Brookings
Kim, Thomas H. ..... Huron
King, Bernard F. ..... Salem
*King, Lydon M., Jr. ... Sioux Falls
Kittelson, H. Otis ..... Sioux Falls
Klar, Werner ..... Fort Meade
Knecht, John F. ..... Martin
Knowles, Roy C. ..... Sioux Falls
Knudson, Donald H. .... Sioux Falls
Knutson, Dennis D. .... Sioux Falls
*Kohlmeyer, Frederick C. Sioux Falls
Kom, Carlton J. ..... Aberdeen
Koob, K. Gene ..... Sioux Falls
*Koren, Paul H. ..... Rapid City
Kortum, Cynthia L. ..... Huron
Kosa, Anna ..... Webster
Kosse, Karl H. ..... Aberdeen
Kovarik, Joseph A. ..... Rapid City
Kovarik, Richard A. .... Rapid City
Kovarik, Wenzel J. ..... Rapid City
Krafka, Thomas L. ..... Rapid City
Kramer, Charles G. Fort Thompson
Kullbom, James B. ..... Rapid City
Kunz, James A. ..... Rapid City
Kurch, Julic Ann ..... Huron
Kwan, Francis P. ..... Rapid City

Lakstigala, Peters E. .... Sioux Falls
Lamb, Marlin ..... Watertown
Lampert, Arthur A., Jr. ... Madison
*Lampert, Arthur A., Sr. Rapid City
Lang, Durward M. ..... Omaha, NE
Langdon, John G. ..... Sioux Falls
Lankhorst, Barry J. ..... Sioux Falls
Lardinois, Clifford C., Sr. ... Huron
Larke, Daryl S. ..... Mich.
Larson, James C. ..... Watertown
Larson, Leland J. ..... Sioux Falls
Larson, Paul M. ..... Watertown
Lauer, David A. ..... Sturgis
*Leander, Richard B. ... Sioux Falls

Lee, Si Gaph ..... Sioux Falls
Lenz, Bernard T. ..... Huron
Leon, Paul R. ..... Aberdeen
Liedtke, Curtis J. ..... Sturgis
Likness, Clark W. ..... Watertown
Lindbloom, Buron O. ..... Pierre
Linde, Leonard M. ..... Mobridge
*Lloyd, John H. ..... Sun City, AZ
Long, Ronald A. ..... Brookings
Looby, Thomas L. ..... Sioux Falls
Loos, Charles M. ..... Rapid City
Loos, Gerald D. ..... Sioux Falls
Lopez, Alberto S. ..... Hot Springs
Lovrien, Fred C. ..... Sioux Falls
Lowery, Gayla S. ..... Sioux Falls
Luechtefeld, Nancy ..... Sioux Falls
Lushbough, Bruce C. .... Brookings
Lyso, Melford B. ..... Yankton

Mabee, Judson O. ..... Mitchell
Mabee, L. M. ..... Sioux Falls
*Mabee, Oscar J. ..... Mitchell
Madison, Dean L. ..... Sioux Falls
Magnuson, Gregory L. .. Sioux Falls
Malm, John A. ..... Gregory
Mangulis, George J. ..... Philip
Manzo-Chua, D. ..... Hot Springs
*Maresh, Everett R. .... Sioux Falls
Margallo, Lucio N., II ..... Mitchell
Mark, Curtis L. ..... Viborg
Markusfeld, Jack M. .... Rapid City
Marquardt, Clark ..... Rosebud
Marschke, Robert F., Jr. Sioux Falls
Marshall, J. B. ..... Rapid City
Massa, Lawrence L. ..... Sturgis
Masterson, Thomas E. .. Sioux Falls
Mattson, William J. ..... Rapid City
Matustik, M. C. ..... Sioux Falls
McClaflin, Richard R. ... Watertown
McFee, John L. ..... Ipswich
McGee, Robert C. ..... Aberdeen
McGrann, James R. .... Sioux Falls
*McGreevy, John V. ... Sioux Falls
McGreevy, Patrick S. ... Sioux Falls
McGuigan, Patrick M. ... Rapid City
McHardy, Bryson R. .... Brookings
McIntosh, George F. ..... Eureka
McKercher, Scott ..... Sioux Falls
McMillin, J. Michael ... Sioux Falls
*McVay, Chester B. ..... Yankton
McVay, Michael R. ..... Yankton
Melvin, Gary ..... Fort Meade
*Merryman, Murlin P. .. Rapid City
Messner, Frank D. ..... Yankton
Meyer, Larry A. ..... Yankton
Meyer, Robert J. ..... Watertown
Michieli, Jose C. ..... Watertown
Millea, Roger P. ..... Rapid City
Mohler, Charles Wm. ... Sioux Falls
Moller, Christopher J. ..... Mitchell
Monfore, James E. ..... Miller
Monson, Charles D. ..... Parkston
Morris, Alan D. ..... Sioux Falls
Mortimer, Sam L. ..... Rapid City
Mueller, Eric H. ..... Tripp
Munson, David P. ..... Sioux Falls
Munson, H. Benjamin ... Rapid City
Mutch, Milton G. ..... Sioux Falls
Myrmoe, Arlin M. ..... Aberdeen

Naughton, Gregory ..... Sioux Falls
Neidich, Gary ..... Sioux Falls
Nelimark, Robert A. .... Sioux Falls
Nelson, Earl G. ..... Viborg
Nelson, Lawrence F. ..... Webster
Nelson, Parry S. ..... Watertown
Nelson, Richard A. ..... Sioux Falls
Nelson, Robert E. ..... Sioux Falls
Nemer, Raymond G. ..... Gregory
Neu, Norman D. ..... Rapid City
Neumayr, Robert J. ..... Yankton
Nice, Richard F. ..... Sioux Falls
Nicholas, George A. ..... Huron
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O'Brien, Peter J. ..... Sioux Falls
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Tschetter, Richard T. ... Sioux Falls
Tschetter, William R. ... Rapid City
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IT SOUNDS FAMILIAR . . . "THAT'S NOT MY JOB!"

This is a story about four people: EVERYBODY, SOMEBODY, ANYBODY, and NOBODY. There was an important job to be done and EVERYBODY was sure that SOMEBODY would do it. ANYBODY could have done it, but NOBODY did it. SOMEBODY got angry about that, because it was EVERYBODY'S job. EVERYBODY thought ANYBODY could do it, but NOBODY realized that EVERYBODY wouldn't do it. It ended up that EVERYBODY blamed SOMEBODY, when NOBODY did what ANYBODY could have done. Author: SOMEONE!

President Reagan launched his campaign with a call for citizen involvement in the social and health problems of the country. Private sector responsibility for the nation and its people was the focus as the President called for a return to volunteerism. While recognizing the great problems besetting our country, let us focus on some of the qualities of our auxiliary which holds the promise of overcoming these ills and clearing the way for still greater advances.

The Legislation Committee motto, "It's Up to You," is a challenge to every auxilian. You have a choice. Continue to think of legislation as something that someone else will take care of, or get involved. Read up on what's happening, help educate others, become involved in a political campaign, establish yourself as a resource person both for candidates and the public. Accept the challenge of involvement!

The AMA Auxiliary urges every member to take "legislative action." Many of our state representatives and senators will be making bids for re-election this year. It is urgent that we let them know our views and concerns.

Still unresolved and coming up before Congress are numerous health legislation issues. They include organ transplants, severely impaired newborns, and protective pricing systems.

Communicate with your legislators! Write, call, or join So Da Pac (South Dakota Political Action Committee). According to the AMA, ". . . PACs are a positive development permitting many persons to participate in our election process." PACs operate entirely from voluntary contributions. PACs monitor legislation, study the voting record, keep us informed, offer an opportunity for our input, and extend support to those candidates who best represent the principles of the medical profession and the interest of those it serves.

VOTER REGISTRATION DRIVE for physicians and members of their families will be launched by the AMA and AMA Auxiliary at the Annual Meeting of the House of Delegates in June (1984). In some areas of the country, as many as 40 per cent of eligible physicians have not registered to vote. The drive will be conducted by the AMA Auxiliary with assistance from state, county, and specialty societies.

The future beckons us — DON'T SIT OUT 1984 — EXERCISE YOUR RIGHT — STAND UP AND BE COUNTED! 350,000 AMA Members and Auxiliary + Spouses, Family, Employees = 1,000,000 Eligible Voters! That's voting power in today's America!!!

"The person who says that he is above politics is really saying democracy is beneath him.

Mary Wrage

Mary Wrage, President
South Dakota State Medical Association Auxiliary

Kenneth Engelhart, M.D., Watertown, has been appointed to the board of St. Ann's Hospital in Watertown.

* * *

Drs. Richard G. Belatti, Sioux Falls, and **William J. Howard,** Rapid City, were elected to three year terms on the South Dakota Lung Association Board.

* * *

The American Board of Family Practice announced that **Dr. Thomas M. Dean,** Wessington Springs, has completed requirement for recertification by the board.

* * *

Loyd Wagner, M.D., Sioux Falls, was elected to a three year term on the Board of Governors of the College of American Pathologists. He is the first South Dakota physician elected to the Board. Dr. Wagner is also the current vice speaker in the CAP House of Delegates.

* * *

The American College of Physicians announced that **Fred C. Lovrien, M.D., Mary Theresa Slattery, M.D.** and **Leonard M. Gutnik, M.D.** have been elected to Fellowship. All three physicians practice in Sioux Falls.

* * *

Robert Rietz, M.D., Brookings, has been appointed to the position of Adjunct Professor of Nursing, South Dakota State University. He is a practicing ear, nose and throat specialist in Brookings.

* * *

Harold L. Aanning, M.D., general surgeon, has joined the Yankton Medical Clinic. Dr. Aanning was born in Norway, he immigrated with his family to the United States in 1947. He graduated from the University of Nebraska College of Medicine in 1976; and completed a five year surgery residency at the University of Nebraska in 1981. He then served two years in the U.S. Navy and was just recently discharged.

Dr. Richard Sample, Madison, has been named a Fellow of the American Academy of Family Physicians.

* * *

Drs. James Gaede, Mitchell and **Jerome Bentz,** Platte, have been named diplomates of the American Board of Family Practice. **Dr. G. Robert Bell,** De-Smet, has been recertified as a diplomate of that Board.

* * *

William W. Quick, M.D., endocrinologist in Yankton, has been elected vice president of the American Association of Diabetes Educators for 1983-84. He previously served as treasurer and chairman of the Budget and Finance Committee for the national organization of health professionals involved in teaching diabetic patients.

* * *

Dr. Robert Seidel, Sioux Falls, and **Dr. John Jones,** Chamberlain, have been named a Fellow of the American Academy of Family Physicians.

* * *

An Aberdeen orthopaedic surgeon, **K. Stephen Kazi, M.D.,** has been named a member of the American Academy of Neurological and Orthopaedic Surgeons.

YOUR CONTRIBUTION IS NEEDED TO THE SOUTH DAKOTA MEDICAL SCHOOL ENDOWMENT FUND

Dr. Charles J. McDonald, Sioux Falls, died February 14 at the age of 76. He was born in 1907 in Sioux Falls. He graduated from Creighton University Medical School in 1933 and served his internship at St. Alexis Hospital in Cleveland, Ohio and his residency in the Cleveland City Hospital. He started his General Practice in Sioux Falls in 1936.

He married Elizabeth Murphy in 1936 in Warren, Ohio.

Dr. McDonald's medical achievements were many. In his 48 year career he served as chief of staff at both Sioux Valley and McKennan Hospitals, was past president of the South Dakota State Medical Association and medical director of South Dakota Blue Shield. He was also physician for Minnehaha County and the South Dakota State Penitentiary. He was active in the Catholic Physicians' Guild, Sioux Valley Medical Society and the South Dakota Chapter of the American Academy of General Practice. He served on the board of directors for the Center for Western Studies and was a member of the local historical and archeological societies. He was also a fourth degree member of the Knights of Columbus and an accomplished amateur magician.

Dr. McDonald's warm sense of humor and his love for a good joke will long be remembered by his colleagues.

Survivors include his wife; four daughters, Mrs. George (Margaret) Swann Jr., Sioux Falls, Mrs. Laurel (Roberta) Mitchell, Sioux City, Ia., Mrs. Louis (Mary) Oswald, Louisville, Ky., and Mrs. Kenneth (Kathleen) Kaufmann, Louisberg, Pa.; 16 grandchildren and one great-grandchild.

Edward A. Rudolph, M.D., Woodland Hills, CA, died May 29, 1983. He was formerly an ophthalmologist in Aberdeen. Dr. Rudolph was born in Alpena, MI. He received his M.D. diploma from the U. of Michigan in 1931 and interned at the U. of Michigan Hospital. He practiced medicine in Michigan and at the Mayo Clinic in Rochester, MN before coming to Aberdeen in 1933 where he practiced medicine until his retirement in 1981. He joined the South Dakota State Medical Association in 1948 and was an honorary member at the time of his death.

Dr. Rudolph's wife, Beulah, still resides in Woodland Hills, CA.

Dr. Vernon H. Cutshall, 63, died in February in Sioux Falls. He was a family practice physician in Sioux Falls since 1951. He was a member of the 7th District Medical Society and the South Dakota State Medical Association. He served on the staffs at McKennan Hospital and Sioux Valley Hospital and was chief of staff from 1959 to 1960.

He was born March 9, 1920 in Palatine, Ill. and grew up in Nebraska. He attended the University of Washington and received his medical degree from the University of Nebraska in 1950. He served his internship at Fitzsimmons Army Hospital, Denver, Colo. in 1951. He was a lieutenant in the U.S. Air Force during World War II and was a B-29 navigator.

He married Margaret Strain in 1942 in Sioux Falls.

Survivors include his wife; two sons, Bruce, Lincoln, Neb. and Don, Dublin, Ireland; five grandchildren and a brother, Dr. Vincent Cutshall, Sioux Falls.

THE SOUTH DAKOTA JOURNAL OF MEDICINE

Journal of Medicine, 608 West Avenue, North, Sioux Falls, SD

Subscription $10.00 per year $1.50 per copy
Foreign $15.00

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ORIGINAL MANUSCRIPTS: Material appearing in all publications of the Journal of Medicine should be typewritten, double-spaced and the original copy, not the carbon should be submitted. An abstract of 100-200 words should accompany each scientific article. Footnotes should conform with the requirements for manuscripts, and each manuscript should include the name of the author, title of article and the location of the author. The used manuscript is not returned but every effort will be made to return manuscripts not accepted or published by the Journal of Medicine. Articles are accepted for publication on condition they are contributed solely to this Journal.

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September

Urology Today, Sheraton Midway Hotel, St. Paul, MN, Sept. 14-15. AAFP & AMA Category I credits available. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

October

Internal Medicine Review, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, Oct. 3-5. Category I credits available. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

1984 ASPRS/PSEF/ASMA Annual Scientific Meeting, Las Vegas, NV, Oct. 9-14. Contact: ASPRS/PSEF Executive Off., 233 N. Michigan Ave., Ste. 1900, Chicago, IL 60601.

Impotence and Penile Implants, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, Oct. 10. Category I credits available. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Group Psychotherapy: The Menninger Foundation, Topeka, KS, Oct. 10-13. Fee: $265. 21 hrs. Category I credits. Contact: Mrs. Jayne Roberts, Div. of CME, The Menninger Foundation, Box 829, Topeka, KS 66601. Phone: (913) 273-7500, ext. 5992.

Adolescent Medicine, Earle Brown Ctr., U. of Minn., St. Paul, MN, Oct. 12-13. Category I credits available. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 272-8012.

Maxillofacial Trauma, Landmark Ctr., St. Paul, MN, Oct. 12-14. Category I credits available. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Colon and Rectal Surgery, Mayo Mem. Aud., U. of Minn., Minncapolis, MN, Oct. 17-20. Category I credits available. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Primary Care Update, MGM Grand Hotel, Las Vegas, NV, Oct. 22-25. Fee: $185. 24 hrs AAFP & AMA Category I credits. Contact: Interstate Postgraduate Medical Asso., Box 1109, Madison, WI 53701. Phone: (608) 257-6781.

Obstetrics and Gynecology Annual Autumn Seminar, Amfac Hotel, Minncapolis, MN, Oct. 24-26. Fee: $295. 17.5 hrs. AAFP & AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Spine Update 1984, Hyatt Regency Hotel, San Francisco, CA, Oct. 24-26. Fee: $295. 18 hrs. Category I credits. Contact: Harry K. Genant, M.D., Prof. of Rad., Ortho. Surg. & Med., U. of Calif. School of Med., San Francisco, CA 94143.

The American Academy of Clinical Anesthesiologists Fall Seminar, Grove Park Inn, Asheville, NC, Oct. 24-27. 11 hrs. Category I credits. Contact: AACA, Box 11691, Knoxville, TN 37939-1691. Phone: (615) 588-6279.

Implant Surgery, Blodgett Mem. Med. Ctr., Grand Rapids, MI, Oct. 25-27. Fee: $375. 18 hrs. Category I Credits. Contact: Am. Soc. for Surg. of the Hand, 3025 S. Parker Rd., Ste. 65, Aurora, CO 80014. Phone: (303) 755-4588.

Third Annual Update: Emergency Medicine for Primary Care Physicians, St. Paul Hotel, St. Paul, MN, Oct. 25-27. AAFP & AMA Category I credits available. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

Infectious Disease in the Elderly, Pills. Aud., Hennepin County Med. Ctr., Minneapolis, MN, Oct. 31. Category I credits available. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

November

Stroke Care Update-1984, Sheraton Midway Hotel, St. Paul, MN, Nov. 2. AAFP & AMA Category I credits available. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

Leukemia Society of America/1st Regional Medical Symposium, Hyatt Regency, New Orleans, LA, Nov. 8-10. Category I credits available. Contact: Leukemia Society of America, 800 Second Ave., New York, NY 10017.

Fall Conference on Anorexia and Bulimia, Barbizon Plaza Hotel, New York, NY, Nov. 17-18. Contact: Center for Study of Anorexia & Bulemia, 1 West 91st St., New York, NY 10024. Phone: (212) 595-3449.

December

Coronary Heart Disease: A Comprehensive Approach to Principles and Practice, Radisson Plaza, St. Paul, MN, Dec. 5-8. AAFP & AMA Category I credits available. Contact: CME, St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

Second Annual Hawkeye Sports Medicine Symposium, Iowa Mem. Union, U. of Iowa, Iowa City, IA, Dec. 6-8. Contact: Deborah Long Hatz, Prog. Ass't., Office of CME, U. of Iowa College of Med., Iowa City, IA 52242.

STACKS

Published Monthly by the S.D. State Medical Assn.

Volume XXXVII September 1984 Number 9

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Clinicopathological Conference Twenty-Seven Year Old Postpartum Gravida IV Para IV With Hypotension and Chest Pain

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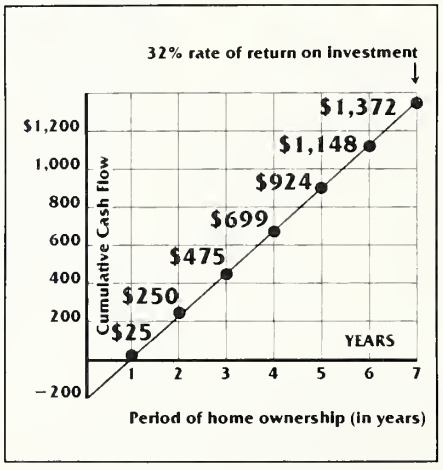

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SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 9 September 1984 (ISSN 0038-3317)

Official Publication of the
South Dakota State
Medical Association

the South Dakota Chapter
of The American Academy
of Family Physicians

the South Dakota Chapter
of the American College
of Surgeons

the South Dakota
Psychiatric Association
of the American
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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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Use of the Epidural Blood Patch in the Treatment of the Post Dural Puncture Headache

Edward F. Anderson, M.D.*

ABSTRACT:

The postdural puncture headache is one of the most common complications of a dural puncture whether it be secondary to a spinal anesthetic, epidural anesthetic or pain block, or diagnostic lumbar puncture or myelography. The history, characteristics, pathophysiology, and various modes of treatment of the postdural puncture headache are described in the article. A history of the various methods used to reduce cerebrospinal fluid leakage, including most recently the epidural blood patch, is presented. The theory, clinical experience, indications and contraindications in the use of the epidural blood patch for the treatment of the postdural puncture are discussed. In general, if conservative treatment of the postdural puncture headache is unsatisfactory after 48 hours, the administration of an epidural blood patch should be considered. Contraindications to its use include: septicemia, localized infection in the area to be injected, blood dyscrasias, use of anticoagulants, and possibly the presence of a neurologic deficit.

One of the most common complications of a dural puncture is a headache. Although it can be secondary to either a spinal anesthetic for surgery and obstetrics or an inadvertent dural puncture during the administration of an epidural in the surgery suite, delivery room or pain clinic, it is more likely to occur after diagnostic lumbar puncture and myelography. Therefore, it is more appropriate to call this a post dural puncture headache (PDPH) rather than spinal headache.[1]

Corning described the first PDPH in 1885.[2] Reflecting on his own postural headache after the attempt to induce spinal anesthesia in 1898, Bier speculated that the escape of a considerable amount of spinal fluid could result in these effects.[3] Sicard, in 1902, and MacRobert in 1918, again postulated that the PDPH was due to leakage of the spinal fluid through the dural puncture.[2]

Not every headache following a spinal or epidural anesthetic or a myelogram is a PDPH. Headaches can be secondary to a myriad of causes especially in the postoperative patient or patient with other pain. Typically, the PDPH has certain characteristics. Its usual onset is not until 24 to 48 hours following the dural puncture. The headache is first noted only when the patient sits upright or stands for brief periods and subsides when reassuming the horizontal position. Indeed, as Bier noted on August 24, 1898: "All these symptoms . . . disappeared as soon as I lay down horizontally, but they returned when I arose."[3] The headache is most commonly occipital in location with radiation into the posterior cervical area and is occasionally associated with nausea, vomiting, diplopia, dizziness, photophobia, tinnitus, or decreased hearing acuity. Occasionally, the headache is described as frontal in location with posterior orbital radiation. It is usually dull and aching in nature.[1] The mean duration of the untreated PDPH is four days and 80% of patients will recover spontaneously within two weeks. Rarely,

* Anesthesiologist and Medical Director, Pain Control Clinic, Sioux Valley Hospital, Sioux Falls, South Dakota.

the PDPH may persist for three to five months.[4]

There have been several series which studied the factors contributing to and the overall incidence of the PDPH. In general, the incidence is greater with larger needles, multiple attempts, and in the third and fourth decades of life. Vandam and Dripps found the incidence of headache after spinal anesthesia for vaginal delivery highest in females age 20-39.[4] Benzon et al found a 17.4% incidence in patients 20-29, 11.1% incidence in 30-39 year old patients and steadily decreasing with advancing age.[5] Many authors reported that the incidence of headache after spinal anesthesia directly correlated with the size of the needle. Vandam and Dripps found the incidence of a 24 gauge needle to be 6% whereas that for a 16 gauge was 18%.[4] Others reported the incidence of PDPH with 26, 25, 24, and 18 gauge needles to be 0.33%, 2.2%, 8%, and 24%.[6, 7, 8, 9] Benzon et al found a 10.7% incidence of PDPH with 22 gauge needles and 5.1% incidence with 25 gauge.[5] (Table I) Crawford found that 70% of 269 patients having an inadvertent dural puncture with an 18 gauge epidural needle developed a PDPH. In the vast majority of his patients, the headache cleared on the 6th postpuncture day with no definitive therapy given.[11] The incidence of PDPH after a diagnostic lumbar puncture varies from 18% to 32% and after a myelogram has been reported between 31% and 44%.[12] After inadvertent puncture during epidural anesthesia the incidence is reported to be 76.9%.[10] The onset was greatest in the first and second postoperative days. The duration was one day to 12 months with 53% resolved in four days, 72% in one week and 80% in two weeks when untreated.[4, 13] Jones found, in a study of 1134 post-spinal patients, that there was no predictable pattern relating to the time spent in recumbency to the occurrence of PDPH, but once the headache occurs, recumbency aids in pain relief.[2]

The pathophysiology of the PDPH is due to loss of cerebrospinal fluid (CSF) through the puncture site with a resultant decrease in pressure in the subarachnoid space.[1] This was confirmed when, in 1943, Kunkle et al produced immediate headaches similar to the PDPH by draining the cerebrospinal fluid from normal human subjects. The headache results because the CSF leakage occurs at a greater rate than CSF production. The leakage rate is determined by the pressure differential across the dura and the dimensions and characteristics of the puncture hole. The vertical position not only increases this pressure differential, but also causes descent of the brain. Resultant traction on the intracranial sinuses, cerebral vessels and tentorium produce position-sensitive referred pain to the head and neck.[14, 15] The needle tract through the dura has been shown to be patent for several days after the lumbar puncture.[16]

Treatment of the PDPH has included such modalities as increased hydration, bed rest, analgesics, caffeine, ethanol, carbon dioxide inhalation and the abdominal binder forcing greater amount of blood into the epidural venous plexus.[1, 14] Most of the methods used to increase CSF production are ineffective. Little evidence exists that an increase in fluid intake will decrease the severity of the PDPH. In fact, the increase in fluids will necessitate increased urination resulting in a worsening of the headache. Instead, methods used to reduce CSF leakage should be attempted to decrease the intensity of the PDPH.[14]

In 1930, Nelson used catgut dural plugs and successfully alleviated the PDPH in 50% of his patients. The technical difficulty with this method and the occurrence of a cauda equina syndrome in 50% of his patients prevented the acceptance of this technique.[16] In 1950, Rice reported on the use of epidural and subarachnoid injection of saline for the treatment of the PDPH.[17] The saline is readily absorbed, however, and the relief may only be temporary.

Gormley, in 1960, mentioned that the incidence of PDPH was lower than expected after bloody spinal taps. He theorized that epidural bleeding from a traumatic dural puncture might lead to clot formation over the dural tear and prevent spinal fluid loss.

TABLE I
INCIDENCE OF PDPH WITH DIFFERENT SIZE NEEDLES

	Size 16	18	19	20	22	24	25	26
Green B.A., 1950[8]				33.3%		8%		.4%
Harris, Harmel, 1953[9]		24%						
Vandam, Dripps, 1956[4]	18%		10%	14%	9%	6%		
Myers, Rosenberg, 1962[6]				23%	10%			.33%
Craft, 1973[10]		76.9%						
Abouleish, 1974[7]							2.2%	
Benzon, 1980[5]					10.7%		5.1%	
Crawford, 1981[11]		70%					15%	

Gormley then reported the cases of seven patients all having immediate relief of their PDPH when having 2-3 ml of autologous blood injected into their epidural spaces.[18] Ozdil, in 1965, reported a 100% success rate in preventing PDPH in surgical patients receiving spinal anesthesia by injecting 2.5 ml of clotted autologous blood into the epidural space as the spinal needle was withdrawn.[19] DiGiovanni, in 1970, published the results of a series of patients receiving an autologous injection of blood into the epidural space and referred to it as the epidural blood patch (EBP).[20]

The technique of the EBP is as follows. The patient is brought to the block room or pain clinic. The technique, objectives and risks of the blood patch are discussed with the patient. Once consent is given, the patient is placed in the lateral position with the hips and knees flexed. The back is scrubbed and prepped with Betadyne or other appropriate antiseptic solution. The skin over the same interspace as the previous dural puncture and underlying subcutaneous tissue are infiltrated with 1% Xylocaine. A 17 or 18 gauge epidural needle (Figure 1A & B) is then inserted through the midline of the interspace into the epidural space. The antecubital fossa of the upper arm is prepped with Betadyne by an assistant. Ten ml's of blood is withdrawn in an aseptic manner and then injected into the epidural space. The patient remains in the supine position for approximately thirty minutes and is then allowed to return to the hospital room or home. Relief from the headache is usually immediate.

The theory of the EBP is that it probably forms a gelatinous tamponade which prevents further leakage of CSF allowing the dura to undergo normal healing. (Figure 2) This technique causes no greater tissue reaction than that which occurs following routine lumbar puncture and less than that following laminectomy.[21]

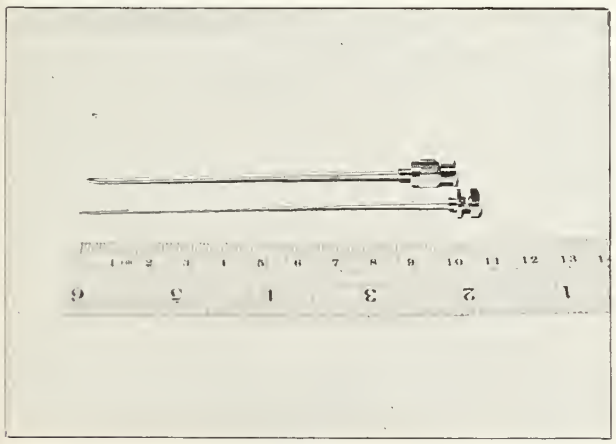

Figure 1A
The Tuohy epidural needle with its stylet removed. A Hustead epidural needle may also be used.

Figure 1B
Close-up of the beveled tip of the Tuohy epidural needle.

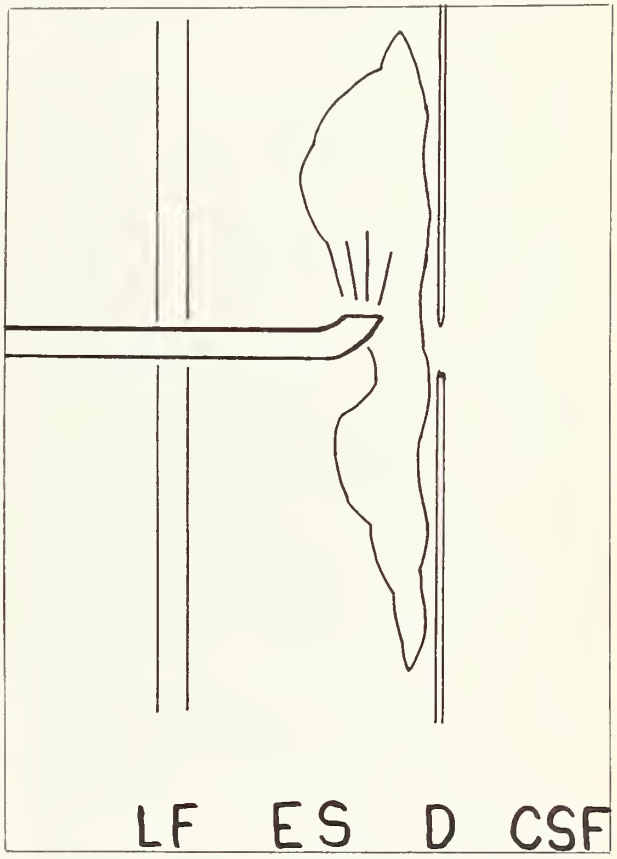

Figure 2
The epidural needle pierces the ligamentum flavum (LF) to enter the epidural space (ES). The injected autologous blood (the epidural blood patch) forms a gelatinous tamponade over the hole in the dura (D) which prevents further leakage of cerebrospinal fluid (CSF).

The results of EBP are outstanding. Many studies (Table II) have been reported demonstrating the effectiveness of this technique. In general, if one waits greater than 24 hours from the dural puncture, a 95% success rate can be achieved. Performing the EBP earlier than 24 hours from the puncture ironi-

TABLE II
SUCCESS RATE OF THE EPIDURAL BLOOD PATCH

	Year	# Pts.	Relief with first EBP	Relief after second EBP
Gormley[18]	1960	7	7 (100%)	
DiGiovanni & Dunbar[20]	1970	45	41 (91.1%)	
DiGiovanni et al[21]	1972	63	61 (96.8%)	
Glass & Kennedy[22]	1972	50	47 (94%)	
DuPont & Sphire[23]	1972	42	40 (95.2%)	1/1
Vandrell & Bernards[24]	1973	60	58 (96.7%)	
Ostheimer et al[25]	1974	185	182 (98.4%)	
Abouleish et al[26]	1975	118	105 (89.0%)	10/11
Khurana & Bali[27]	1978	125	117 (93.6%)	
Loeser et al[28]	1978	66	42 (63.6%)*	
Azar & Betcher[29]	1980	8	7 (87.5%)	
Crawford 1980, 1981[11, 30]	1981	173	165 (95.3%)	
		942	872 (92.6%)	

* Loeser et al evaluated the time versus success rate for EBP and found that if the EBP were done before 24 hours had elapsed following the dural puncture, the success was only 29%. If done after 24 hours, the success was 96%. If this study is eliminated from the results, the overall relief is 830/876 patients or *94.7%*.

cally has a low success rate.[28] There is an occasional skeptic concerning the efficacy of this technique claiming the PDPH will spontaneously resolve in 4-5 days.[31] However, this has been refuted by the many studies previously mentioned.

In the occasional patient when the headache is not relieved with the first block, reblocking has at least the same rate of success (i.e., 95%).[26] The amount of autologous blood injected is recommended from 2.5 ml (Gormley) to 20 ml (Crawford).[11, 18, 30] Most authors accept 10 ml. According to Palahniuk and Cumming, the prophylactic administration of an EBP after spinal anesthesia does not prevent PDPH.[32] On the other hand, Ozdil in 1965, as previously mentioned, prevented the development of a PDPH in all his surgical patients with a prophylactic EBP.[19] Gutterman and Bezier also found that the prophylactic administration of 2.5 ml of autologous blood while withdrawing an 18 gauge spinal needle during myelography reduced their incidence of PDPH from a control of 39% to 5%. The 5/100 patients who did develop a PDPH had much less intense headaches.[12] Milette et al have reported their successful use of the EBP in the treatment of the post-myelogram headache.[33]

One of the concerns regarding the use of the EBP is the ability to administer an epidural anesthetic in the future. The reasoning is of the possible subsequent obliteration of the epidural space following organization of the blood clot. The resulting possible adhesion between the dura and ligamentum flavum following EBP could lead to future inadvertent dural puncture or else prevent the spread of local anesthetic in the epidural space. Two reports have concluded that the EBP does not obliterate the epidural space and should not preclude the use of a regional block for later surgical or obstetric procedures.[34, 35]

Rare complaints have followed the use of this block including: transient paresthesis in the lower extremities, meningismus, abdominal cramping, tinnitus, and vertigo during the blood injection. Some patients have had an increase in temperature lasting several hours.[34] Mild backache at the injection site lasting 48 hours may occur.[26]

A case report of lumbosacral meningismus was felt to be secondary to inadvertent injection of the blood into the subarachnoid space.[36] One patient had a transient episode of radicular back pain to the right buttock which resolved in two weeks. Possible explanations are that the clot began to retract causing traction between the nerve roots and surrounding structures or that the clot resulted in mechanical compression of the nerve roots when the patient flexed her spine.[37] There was an additional report of the occurrence of a spinal subdural epiarachnoid hematoma in one patient. This was unusual because six EBP's were performed including three in which the dura was inadvertently punctured.[38] It is the opinion of Barros that no greater than two EBP's are necessary on any given patient.[39] The occurrence of an epidural hematoma is possible but very unlikely. If an inadvertent puncture of an epidural vein during the EBP attempt occurs, the injection should not be carried out since the bleeding will form a clot over the puncture.[40]

Two theoretical, but as yet unreported complications after an EBP, are epidural abscess formation and adhesive arachnoiditis. Generally, if one is careful in the selection of patient with a PDPH for EBP and follows scrupulous technique, the above complications can be avoided. Contraindications to the use of the EBP are: septicemia, localized infection or

inflammation in the area to be injected, patients with blood dyscrasias or taking anticoagulants, and possibly the presence of a neurologic deficit.[21]

In conclusion, the use of the EBP as a treatment of the PDPH has been reported. With an unremitting PDPH conservative management such as bed rest, hydration, and analgesics should be attempted first. If the above treatment is unsatisfactory after 48 hours, the EBP should be considered. The low morbidity and 95% immediate success rate on first injection makes the EBP an excellent alternative in the management of the PDPH.

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Isn't it interesting that our presidential elections and the olympic games coincide every four years? 1984 is our year to "Go for the Gold" and exercise our privilege of casting our vote November 6.

This year the AMA Auxiliary through each state and district is promoting a voter registration program. It has been carefully documented that in some areas of our country as many as 40% of eligible physicians have not registered to vote. I am sure South Dakota is not in this category. However, we should be 100% registered. An active program is being promoted by our SDSMA Auxiliary with Mary Wrage, our state president, acting as ramrod. This is a non-partisan program that will be carried out from the "grass roots" through each district. Our auxiliary has been a strong arm of our association and we must continue to support their programs. My personal thanks to all our national, state and district officers of the auxiliary.

Let's all be sure to be registered and then — VOTE!

Howard L. Saylor, Jr., M.D., President
South Dakota State Medical Association

John J. Jacobsen, M.D., Yankton, was elected to fellowship in the American Academy of Pediatrics.

* * * *

Dr. J. Michael McMillan, Sioux Falls, has been named as the president of the American Diabetes Association, South Dakota Affiliate, Inc. Dr. McMillin has been a member of the state board of directors since 1978 and has been vice president of the association since 1982.

* * * *

The following physicians have completed continuing education requirements to retain active membership in the American Academy of Family Physicians: **Drs. Michael Crandall,** Chamberlain; **Forrest Brady,** Spearfish; **Susan Provow,** Vermillion; **Robert Shaskey,** Brookings; **Charles Pelton,** Aberdeen and **Alfred Shousha,** Britton.

* * * *

Yankton physician, **Theodore H. Sattler, M.D.,** has been named one of six South Dakota winners of the 1984 Jefferson Award, presented in cooperation with the American Institute for Public Service.

The award recognizes individuals who are doing outstanding public service benefitting local communities. Dr. Sattler was cited for his contributions in the area of health care.

* * * *

Douglas M. Traub, M.D., Watertown, has been notified of his certification as a diplomate of the American Board of Internal Medicine.

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WELCOME TO CHICAGO!

Welcome to one of the world's most exciting cities. And welcome to the 61st Annual Convention of the American Medical Association Auxiliary (June 17-June 20, 1984). It seems attending meetings and conventions is a major part of our "professional" lives today.

In exemplary fashion, we were given full, extensive coverage in areas of AMA-ERF, Health Projects, Membership, and Legislation during these "Action-Packed Days."

During a colorful celebration of national pride, I was seated on stage with the other state presidents at the opening meeting of the House of Delegates. Following the Presentation of State Presidents, we witnessed the Presentation of Colors by the United States Army Color Guard, Fort Sheridan, Illinois — the National Anthem, Pledge of Allegiance to the Flag, Invocation/Memorial, and the Pledge of Loyalty to the American Medical Association Auxiliary. We listened to Glenda (John) Bates' Report of the President, along with special Keynote Speaker, the Honorable Newt Gingrich, United States House of Representatives (R-Georgia).

A new city, a new hotel, new people to meet — all add up to a stimulating environment away from the routine of our normal lives, as was the case with the South Dakota participants: Marie (James) Hovland, Ila (Bruce) Lushbough, Shirley (James) Ryan, and myself. Marie Hovland is serving on the National American Medical Association Auxiliary Legislative Board and Ila Lushbough is serving as a National Director for the North Central Region of the American Medical Association Auxiliary.

Are you a member of our "Auxiliary A-Team?" Our "Team" is something you should definitely observe, or even participate in!

Compliments of the American Medical Association, I flew the "Friendly Skies" in a DC9 to Washington DC and attended a national training seminar July 11, 1984 at the Dirksen Senate Building and heard details of an important new program that will affect physicians and their spouses in the coming months. Called MEDVOTE, the nonpartisan program is a national voter registration drive for the 1984 elections. MEDVOTE comes on the heels of the AMA's successful "Participation '84" program, initiated in 1983 to encourage and assist physicians in running as delegates and alternate delegates to the 1984 national party conventions. Each District Auxiliary Legislative Chairman is the designated individual to organize the effort on a local level with the completion date of October 1, 1984. (A detailed report will be furnished upon completion.)

The importance of participation in the nation's democratic process is, of course, universally known and acknowledged. Too often, however, physicians and others give only lip service to the matter.

In the upcoming election, American voters will choose a President, 33 senators, the entire House of Representatives, 13 governors, and thousands of state and local officials. The selection of these officials at all levels of government is crucial. Congressional and state legislative interest in health care issues is continuing to increase, and there is talk that 1985 will be the "year of the physician" in Washington.

MEDVOTE has the potential to help assure that the nation has the sound, responsive government to which the American Medical Association is dedicated.

Mary Wrage

Mary Wrage, President
South Dakota State Medical Association Auxiliary

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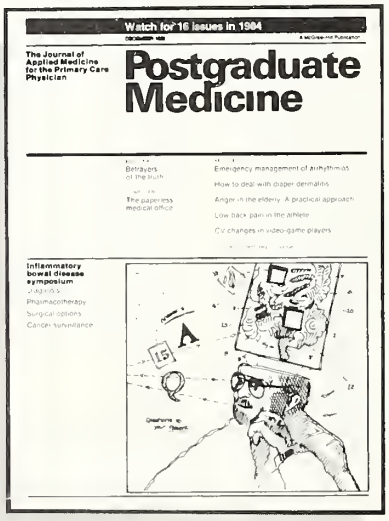

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Twenty-Seven Year Old Postpartum Gravida IV Para IV With Hypotension and Chest Pain

Kenneth M. Johnson, M.D.*
Paul L. Carpenter, M.D.†
Thomas Gaeckle, M.D.‡
Discussers

John F. Barlow, M.D.§
Editor

Case #1022063 (A83-81)

This 27-year old Caucasian gravida IV para IV female was admitted to Sioux Valley Hospital two days postpartum for hypotension and chest pain.

The patient had been healthy with no serious illnesses or hospitalizations other than her pregnancies. There were two other living healthy children. Seven years previously she had had a stillborn baby and had had a very swollen leg for six weeks after that delivery. There was no associated shortness of breath or hemoptysis with this episode and the patient had never had any limitation of activities or cardiac difficulties. There was no other history of previous chest pain, rheumatic fever or other cardiac conditions. During the present pregnancy, the patient had no problems. The delivery was rapid within two hours. During that delivery the patient did have central chest pain during the hyperventilation of labor. In retrospect, the patient did complain of slight pain in the right leg before and after the delivery. One day after the delivery the patient went to the bathroom and felt peculiar and complained of recurrent central chest pain. She returned to bed where she was reported to have had a generalized seizure. She became hypotensive but an electrocardiogram showed no abnormality. Pressor drugs were necessary to raise the blood pressure and she started to complain of more severe central chest pain which radiated down the left arm and was increased on respiration. A lung scan was performed which showed a perfusion defect of the right upper lobe. Laboratory studies reported an elevated prothrombin time and partial thromboplastin time. Because of continued hypotension in the range of 60 systolic, the patient was transferred. During transport she had a short seizure.

There was no other significant history except for congenital abnormalities of the toes and feet.

PHYSICAL EXAMINATION: Pulse 132/min and regular, respiration 36/min and regular, blood pressure 96 systolic and 66 diastolic, height 5′3″ (168 cm), weight 71 kg. The patient was alert, cooperative and oriented. Examination of the head and neck was unremarkable. There were no abnormalities of the thyroid and the neck veins were not distended. The chest was clear to auscultation and percussion. The heart had a regular rhythm with no murmurs or gallops. There were no masses in the breast. The abdomen was nontender and showed no abnormality except for a postpartum uterus. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis yellow, hazy, specific gravity 1.015, pH 6.5, negative for protein, glucose, ketone bodies, bilirubin, or hemoglobin; sediment 2 to 4 leukocytes per high power field and 25-30 red cells per high power field. There were rare squamous epithelial cells and a few bacteria. Hemoglobin 10.4 gm/dl, hematocrit 31% with normal red cells indices, leukocyte count 13.4 K/μl with a differential of 90% segmented neutrophils, 3% neutrophilic bands, 6% lymphocytes and 1% monocytes. The red cells were normocytic normochromic on smear. A platelet count was 170 K/μl. Erythrocyte sedimentation rate was 38 mm/hour; a fluorescent antinuclear antibody test (FANA) was negative. A bleeding time was 6.5 minutes (normal), a prothrombin time was 12.8 seconds with a control of 12 seconds, a partial thromboplastin time 55 seconds with a control of 26 seconds (the latter was slightly elevated), fibrinogen level 385 mg/dl (normal); protamine sulfate test negative. Split products were not elevated on initial determinations but

* Resident in Family and Community Medicine, USD Affiliated program. Sioux Falls, SD.
† Specialist in Cardiology and Internal Medicine, Sioux Valley Hospital and North Central Heart, Clinical Faculty, School of Medicine, University of South Dakota, Sioux Falls, SD.
‡ Specialist in Internal Medicine, Sioux Valley Hospital Clinical Faculty, School of Medicine, University of South Dakota, Sioux Falls, SD.
§ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital: Professor of Pathology, School of Medicine, University of South Dakota, Sioux Falls, SD.

became elevated slightly subsequently. A lactic dehydrogenase (LD, LDH) was 477 IU/L (0-270 IU/L), total protein was 4.6 gm/dl (normal 5.5 to 8.3 gm/dl), calcium 7.5 mg/dl (normal 8.4-10.7 mg/dl), glucose 183 mg/dl (normal 60-125 mg/dl). Alkaline phosphatase, aspartate aminotransferase (AST, SGOT), total bilirubin, phosphorus, blood urea nitrogen, creatinine, uric acid, cholesterol, thyroxine (T4), thyrotropin (TSH), and rheumatoid arthritis tests were negative. Admission sodium was 132 meq/l, potassium 3.7 meq/l, chloride 112 meq/l and CO2 content 15 mm/l. Multiple repeat determinations for sodium and potassium were within normal limits. Admission blood gases revealed a pH of 7.40, PCO2 29 torr, total CO2 content 19 mm/L, PO2 135 torr, oxygen saturation 99%. The patient was receiving oxygen by mask. A urine culture and three blood cultures revealed no growth. Admission creatine kinase values were elevated at 401 and 737 IU/L (normal 18-80 IU/L) with elevated MB (specific for myocardium) fractions at 17% and 14%. LDH isoenzymes revealed an elevated LDH 1 fraction over LDH 2 fraction consistent with myocardial damage, renal cortical damage, or hemolysis. Total LDH was 577 IU/L. Initial electrocardiogram showed sinus tachycardia but no abnormalities. Subsequent electrocardiograms were interpreted as either abnormalities suggesting pericarditis or an acute injury pattern. An initial chest film showed a prominent mediastinal shadow to the right possibly a dilated azygous vein. A computer tomogram was nondiagnostic. An emergency arteriogram of the aortic arch was negative for a dissecting aneurysm. A sector echocardiogram revealed moderate anterior and posterior pericardial effusion. A repeat perfusion lung scan was considered low probability for pulmonary embolism.

A neurologic consultation suggested that the patient's seizures were secondary to hypotension. A Swan-Ganz catheter was passed. A median right atrial pressure was 22 mm of mercury, the pulmonary artery pressure was 30/18 with a mean of 24 mm of mercury, and the mean wedge pressure was 23 mm of mercury. These results were felt to be due to the restrictive effect of pericardial effusion. The patient was started on corticosteroids, antibiotics and heparin with a possible diagnosis of pulmonary embolism. On the fourth hospital day, the patient's fibrin split products were 40 mg/dl and the platelet count dropped to 118 K/UL. The patient developed a mild hypoxia and had episodes of ventricular tachycardia and two episodes of ventricular fibrillation requiring bretylium. By the fourth hospital day the patient's electrocardiogram was showing evidence suggesting acute myocardial infarction and the creatine kinase (CK,CPK) level was 1685 U/L with 17% MB band. Late in the fourth hospital day the patient became increasingly hypotensive and attempts at pericardiocentesis revealed only clotted blood. The patient died on the fourth hospital day.

DR. JOHNSON: I would like to list the pertinent findings in this case:

1. The patient was a 27-year old postpartum female with chest pain.
2. There was a history of swelling of the leg in the postpartum period 7 years previously.
3. On the first day after the delivery, the patient had a seizure, was noted to be hypotensive and required pressors for blood pressure support, but had a normal electrocardiogram.
4. The patient did have some chest pain during labor but this was thought to be insignificant.
5. A ventilation perfusion scan showed a right upper lobe perfusion defect.
6. After referral, the patient had shortness of breath but a repeat ventilation and perfusion scan was considered to show a low probability of pulmonary embolism.
7. Routine studies such as urinalysis, complete blood count with hemoglobin, and platelets were not helpful and were consistent with a postpartum examination.
8. The patient did have an elevated partial thromboplastin time when she was admitted to Sioux Valley Hospital, but I wonder if she was on heparin by this time.
9. The arterial blood gases showed a mild corrected acidosis and was receiving oxygen.
10. After admission, the patient had an elevated CK with elevation of the MB fraction; and, in addition, the LDH was elevated with the isoenzyme fraction 1 being greater than 2 suggesting myocardial damage, renal damage or hemolysis.
11. The electrocardiogram revealed sinus tachycardia, then a picture of pericarditis, and finally a picture suggesting anterior myocardial infarction.
12. The chest x-ray showed a prominent right hilum.
13. An aortic arch angiogram revealed no evidence of dissection of the aorta.
14. Late in the course, the patient developed thrombocytopenia with increasing fibrin split products consistent with disseminated intravascular coagulation secondary to shock.
15. There was a definite diagnosis by sector echocardiogram and electrocardiogram of an acute myocardial infarction with pericardial effusion made before death.

With these facts in mind, let us first discuss the differential diagnosis of chest pain as modified from Braunwald's text book.[21] (Table I)

Pulmonary embolism was considered. The chest pain due to pulmonary embolism is usually secondary to infarction of the lung which is usually manifested on chest x-ray. We did not see evidence of a pulmonary infarction on chest x-ray. The only abnormality was a prominent azygos vein. When the ventilation perfusion scan was repeated, it showed a low probability of a pulmonary embolism. I doubt that it could have become normal so quickly after a known thrombotic occlusion of a pulmonary artery although I cannot explain the previous abnormal scan. She did have shortness of breath, but she had no findings suggestive of deep vein thrombosis and the electrocardiogram showed no right ventricular strain pattern. For these reasons, I feel the diagnosis

TABLE I
CAUSES OF CHEST PAIN

I. NONCARDIOVASCULAR DISORDERS
  A. NEUROMUSCULAR MECHANISM
    Costochondritis and other chest wall syndromes
    Radicular syndromes
    Inflammatory syndromes of the shoulder joint
  B. GASTROINTESTINAL MECHANISM
    Hiatal hernia
    Esophageal disease
    Peptic ulcer or gastritis
    Gallbladder disease
  C. PSYCHOLOGICAL MECHANISM
    Psychoneuroses
    Psychosomatic complaints
II. CARDIOVASCULAR AND PULMONARY DISORDERS
  Coronary artery disease (atherosclerotic versus nonatherosclerotic disease)
  Aortic valve disease
  Idiopathic hypertrophic subaortic stenosis
  Primary myocardial disease
  Pericarditis
  Dissecting aortic aneurysm
  Mitral valve prolapse syndrome
  Pulmonary embolus/infarction
  Pulmonary hypertension, right ventricular strain, or both
  Pneumothorax
  Pleuritis
  Intrathoracic tumor
  Chest pain with normal coronary arteriograms

of pulmonary embolism is unlikely.

One has to consider dissection of the aorta because this occurs in relatively higher frequency in pregnant women compared to women of the same age group who are not pregnant. However, an arteriogram of the aortic arch was completely negative.

We have good evidence that the patient had a myocardial infarction. There was no evidence of this at first but the increased total CK, LD, and isoenzyme patterns and progressively abnormal electrocardiogram are all compatable with an evolving myocardial infarction. I can only sympathize with the referring and attending physicians making a diagnosis of this entity in a young woman.

Wei[22] discusses myocardial infarction in young women in a review of 19 women under 36 years of age who had myocardial infarction and compares them to a group of men of similar age. He found that the men had coronary atherosclerosis usually involving multiple vessels. In the women he found little evidence of disease on the coronary angiograms or postmortem and they had significantly less risk factors for coronary atherosclerosis than the men. He concluded that young males who have myocardial infarction usually have coronary atherosclerosis but in young women nonatherogenic causes of myocardial infarction are more likely. I will include in my discussion at this point causes of myocardial infarction without atherosclerosis. (Table II)

TABLE II
MYOCARDIAL INFARCTION IN ABSENCE OF CORONARY ATHEROSCLEROSIS

CORONARY ARTERITIS
  Syphilitic
  Granulomatous (Takayasu disease)
  Infections and "nonspecific"
  Polyarteritis nodosa
  Disseminated lupus erythematosus
  Rheumatoid arthritis
  Rheumatic fever
  Kawasaki's disease (mucocutaneous lymph node syndrome)
  Ankylosing spondylitis
  Dissection of the aorta
  Dissection of the coronary artery
  Spasm of coronary arteries (Prinzmetal angina with normal coronary arteries)
  Spasm after nitroglycerin withdrawal
TRAUMA TO CORONARY ARTERIES
  Laceration
  Thrombosis
  Iatrogenic
  Coronary artery fibrosis secondary to radiation
CORONARY MURAL THICKENING WITH METABOLIC DISEASES OR INTIMAL PROLIFERATIVE DISEASE
  Mucopolysaccharidoses (Hurler disease)
  Homocystinuria
  Fabry's disease
  Amyloidosis
  Juvenile intimal sclerosis (idiopathic arterial calcification of infancy)
  Intimal hyperplasia associated with contraceptive steroids or with the postpartum period
  Pseudoxanthoma elasticum
CORONARY ARTERY EMBOLISM
  Infective endocarditis
  Thrombus from left atrium, left ventricle
  Prosthetic valve emboli
  Prolapse of mitral valve
  Cardiac myxoma
  Associated with cardiopulmonary bypass surgery and coronary arteriography
  Paradoxical emboli
  Papillary fibroelastoma of the aortic valve ("fixed embolus")
CONGENITAL CORONARY ARTERY ANOMALIES
  Anomalous origin of left coronary from pulmonary artery
  Left coronary artery from anterior sinus of Valsalva
  Coronary arteriovenous and arteriocameral fistulae
  Coronary artery aneurysms
MYOCARDIAL ISCHEMIA DUE TO OXYGEN DEMAND EXCEEDING SUPPLY
  Aortic stenosis, all forms
  Incomplete differentiation of the aortic valve
  Aortic insufficiency
  Carbon monoxide poisoning
  Thyrotoxicosis
  Prolonged hypotension
HEMATOLOGICAL (IN-SITU THROMBOSIS)
  Polycythemia vera
  Thrombocytosis
  Disseminated intravascular coagulation
  Hypercoagulability
MISCELLANEOUS CAUSES
  Myocardial contusion
  Myocardial infarction with normal coronary arteries

Beary et al[16] discusses postpartum myocardial infarctions. He discusses two cases and reviews nine other cases from the literature. In these eleven cases postpartum infarction most often occurred in women in their twenties in a pregnancy complicated by the preeclampsia-eclampsia syndrome and was associated with a high mortality rate. In two patients there were normal coronary artery angiograms. He suggested coronary spasm as a cause of the myocardial infarction in these cases.

The patient could have had coronary spasm which has been known to have been induced by ergonovine, methacholine or immersing of the hands in cold water. This can be reversed by nitrates. There is no record of the patient receiving ergot derivatives in this case although it is a common obstetric practice.

Finally, I would like to conclude my discussion with mention of coronary artery dissection. I could find only 46 reports of this entity. Seventy-five cases were in women and 12 of these were within 12 weeks postpartum. With the exception of three, all of these were diagnosed at autopsy. The left main, left anterior descending or left circumflex coronary arteries were the most common arteries affected. With rare exceptions these patients had normal blood pressures and uncomplicated pregnancies and deliveries. It has been suggested that the effort of straining during labor and delivery may initiate intimal tears with subsequent hemorrhage into the media several days or weeks later. Indeed, some patients have had findings suggestive of a two stage event. Another pathogenetic possibility is a rupture of the vasa vasorum into the media initiating a dissection of the coronary artery wall. The currently popular belief is the degeneration of the ground substance of the medial layer of the arteries commonly seen during pregnancy is instrumental in weakening the vessel wall and producing dissection.

Dr. Johnson's Diagnosis

1. **Acute myocardial infarction.**
2. **Coronary spasm or coronary artery dissection.**

DR. CARPENTER: This patient was a puzzle to us from the very beginning. I must admit that we felt with a history of chest pain, hypotension, leg pain and previous history of leg swelling in the postpartum period associated with an abnormal lung scan, our first thought was pulmonary embolism. The patient was, as you indicated, on heparin therapy by the time she got to this hospital. By the second hospital day after we had received a report of a normal lung scan and had performed a sector echocardiogram which demonstrated a pericardial effusion, we began to seriously doubt our original diagnosis. The electrocardiogram subsequently changed from a pattern of pericarditis to anterior myocardial infarction. We wondered about thrombosis of the coronary artery perhaps secondary to amniotic fluid embolism as a cause of the myocardial infarction.

DR. GAECKLE: I was also perplexed about this case. I thought about coronary artery vasculitis and perhaps a complication of eclampsia.

DR. BRUCE VOGT*: Did the patient receive ergot at the original hospital?

DR. CARPENTER: We don't think so.

DR BARLOW: the patient at autopsy was a well developed, well nourished female with no external features of Marfan's syndrome. In fact, there was loss of both fingers and toes. The pericardium was distended with an estimated 400-500 cc of clotted blood. The heart weighed 325 gm and had a smooth epicardium except for a shaggy 1.2 cm in length laceration of the left ventricle laterally near the atrioventricular sulcus (Fig. 1). The coronary arteries showed only a rare small atherosclerotic plaque but the left main coronary artery was completely occluded by dark red material starting 2 cm beyond the coronary ostium in the aorta. The occlusive process extended into the left anterior descending branch well down the anterior sulcus and involved the proximal circumflex coronary artery. There were two

Figure 1
Hemorrhagic myocardial infarction with tear in myocardial wall which led to hemopericardium and pericardial tamponade.

* Clinical faculty of Family Practice Residency Program, affiliated with School of Medicine, University of South Dakota, Sioux Falls, SD.

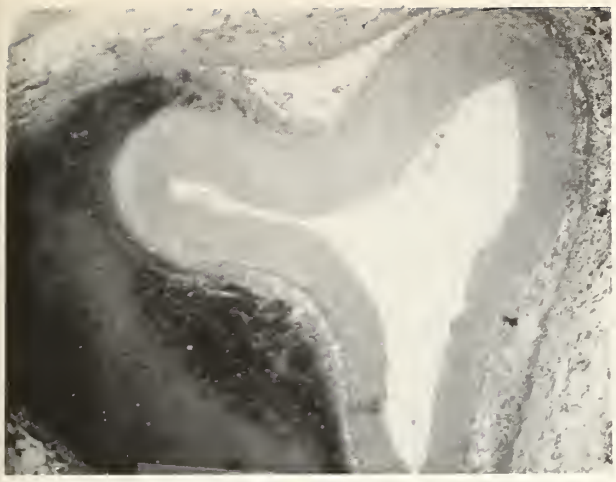

Figure 2
Dissecting channel in outer media of coronary artery. The blood in the new channel compresses and distorts the inner wall and lumen leading to coronary artery occlusion.

separate areas of hemorrhagic softening in the myocardium. One involved the interventricular septum and measured 3 x 2 x 1.3 cm and a second separate area was in the lateral ventricular wall and measured 2 x 1.5 x 1.3 cm. This contained the laceration previously described producing a transmural rupture.

Microscopic examination of these areas revealed hemorrhagic necrosis. The striking finding was in the microscopic examination of the coronary arteries which showed a dissection in the outer media with compression and distortion of the inner media and intima markedly narrowing the lumina. The dissection was completely circumferential in some areas and partly in others (Fig. 2). A focal infiltrate of segmented neutrophils and rare histiocytes was seen in the adventitia. Alcian blue stains revealed increased amounts of material staining similar to mucopolysaccharides in the media of the coronary vessels and aorta. There were small pools of mucin but it was not true mediocystic necrosis and may be the process described in normal pregnancies. There was no dissection of the aortic media found after careful search.

FINAL ANATOMIC DIAGNOSIS

1. **ISOLATED DISSECTING ANEURYSM OR HEMATOMA OF LEFT MAIN CORONARY ARTERY AND ITS BRANCHES.**
2. **MYOCARDIAL INFARCTION, ACUTE INTERVENTRICULAR SEPTUM.**
3. **MYOCARDIAL INFARCTION, ACUTE LATERAL WALL OF LEFT VENTRICLE WITH RUPTURE.**
4. **HEMOPERICARDIUM, MASSIVE.**

DISCUSSION

Dissecting aneurysms of the coronary artery are most commonly secondary to extension from a dissecting aneurysm or hematoma of the aorta. Isolated dissection of major branches of the aorta without aortic involvement has been described most commonly in coronary, renal and carotid arteries. Such dissection may be spontaneous but has also been described after chest trauma such as closed or open cardiac resuscitation and getting kicked in the chest by a horse. Catheterization of coronary arteries for angiography or catheterization or manipulation during aortic and mitral valve replacement or coronary artery bypass surgery are procedures associated with iatrogenic isolated dissection of the coronary arteries.

Spontaneous isolated dissection of a coronary artery is clearly associated with the peripartum state and occurs usually within 80 days of parturition. The first isolated coronary dissection was described in 1931 by Pretty but several series have been described.[1-5] Smith discussed 43 cases. Thirty-two occurred in women and eleven in men — a 3 to 1 female to male ratio. Only two black women were noted; the rest were white. The average age for females was 39 years with an age range of 17 to 67 years. Ten were pregnant and ten were postpartum. The average heart weight was only 324 gm. indicating absence of chronic heart disease. In men the average age was 50 years with an age range of 21 to 63 years. The heart weights averaged 337 gm. In 37 of 43 cases a single vessel was involved and in 30 of those 37 the single vessel was the left anterior descending coronary artery. The right coronary artery was the single artery in 6 cases but the circumflex coronary artery was involved only once. There were 6 cases in which multiple vessels were involved and the left anterior descending was involved in all of these instances. The dissection usually starts 2-4 cm from the aortic ostium and can be of a variable distance but was only 2-4 cm in average length. The dissection usually occurs in the outer media. The false channel fills with blood compressing the lumen and producing occlusion. Twenty-seven of 43 cases caused sudden death with only 4 of the 27 cases having a myocardial infarction demonstrated at autopsy. In 15 cases with documented myocardial infarction most died in one to several days. However, coronary artery dissection has been documented as a cause of an old posterior myocardial infarction in a 40 year old man who died of a fresh anterior myocardial infarction secondary to a recurrent dissection of the anterior descending coronary artery.[4] The author pointed out the very important point that the artery leading to the old infarction appeared normal grossly until histologic sections revealed evidence of the old

dissection. I believe this may be of significance in a time when coronary spasm is blamed for "normal" coronary arteries on angiogram in the presence of clinical ischemic disease of the myocardium.

The causes of dissection are unclear. Although segmented neutrophils, lymphocytes and eosinophils have been described in the adventitia, it is believed that the presence and nature of the inflammatory response is more a function of the time from dissection to death rather than an indication of an arteritic causal factor. Intimal tears are unusual except in iatrogenic cases but have been described in spontaneously occurring cases. The cases occurring during pregnancy are unassociated with pregnancy or atherosclerosis. Cystic medial necrosis has been described in 13 of 43 cases but the patients were not described as having the features of Marfan's syndrome. Some have suggested that the general loosening of connective tissue seen in pregnancy as may be produced in guinea pigs by estrogen or relaxin or the increased mucopolysaccharides seen in the aorta and major arteries in pregnancy predispose to dissection which is initiated by the stress of labor. Thrombi are rarely seen in the true or false lumina. Interesting, only one case of 43 has been diagnosed antemortem.[3] Chronic pulmonary disease has been associated with some but not all cases. Especially the peripartum cases have given little evidence of serious pulmonary disease. From investigation of some of the iatrogenic cases, it has been appreciated that not all cases die. Oral contraceptives have only been implicated in one case[14] and copper deficiency has been suggested as a cause.[13]

It might be important to note some differences between isolated coronary artery dissection and dissecting aneurysm of the aorta.[9] (Table III)

Finally I would like to note that in a time when coronary spasm is a popular theory for ischemic myocardial disease in the absence of severe atherosclerosis, I would like to list the causes of coronary artery occlusion (Table IV)[5] which may cause myocardial ischemia with or without sudden death.

TABLE III

	CORONARY ARTERY (ALONE)	AORTA
Age	Young	Older
Sex	Female predominance	Male predominance
Hypertension	Rare	Common
Peripartum state	Frequent	Rare
Left ventricular hypertrophy	Rare	Common
Atherosclerosis	Mild or none	Moderate
Luminal narrowing	Common	Rare
External rupture	Rare	Common
Chronic state	Probably rare	Well described
Intimal tear	Rare	Common

TABLE IV

CAUSES OF CORONARY ARTERY OCCLUSION

1. **Severe generalized coronary atherosclerosis (most common).**
2. **Acute coronary thrombosis (usually in vessels with an old plaque).**
3. **Hemorrhage into a plaque.**
4. **Coronary artery embolism not associated with significant atherosclerosis**
   a. **infective endocarditis.**
   b. **cardioversion**
   c. **prosthetic heart valve**
   d. **intracardiac thrombus**
5. **Medial dissection of coronary artery**
   a. **Secondary to aortic dissection**
   b. **Primary**

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12 Most-Asked Questions about The Family Health Foundation of America

1. **How did it all begin?**

A. The Family Health Foundation of America was founded in 1958 by the American Academy of Family Physicians. Academy members knew that such a freestanding foundation was vital.

2. **What did they want the Foundation to do?**

A. To improve the health care of the American public.

3. **What happened next?**

A. In the 1960s and early 1970s the FHFA held conferences to spark the budding family practice movement. FHFA helped the American Board of Family Practice get started on a sound footing and, through "seed grants," helped to establish some of the first family practice residency programs.

4. **What is FHFA's relationship to the American Academy of Family Physicians?**

A. FHFA is the Foundation of the American Academy of Family Physicians and serves as the philanthropic arm of the family practice movement. Organizationally, the Board of Directors of AAFP serve as the voting members of the Foundation and are responsible for electing the Board of Trustees and monitoring the activities of the Foundation.

5. **What is FHFA's unique status?**

A. As a "non-private" foundation it can seek grant funds from "private" foundations and corporations.

6. **Has this search been successful?**

A. Very successful, with grant funding from the W. K. Kellogg Foundation exceeding $1,000,000, funding from the Merck Co. Foundation and contributions from 15 pharmaceutical companies providing support for various FHFA programs.

7. **What are FHFA's current interests?**

A. As a grantmaking Foundation, FHFA will carefully utilize the assets it has and will accumulate to:

- Support activities which encourage medical students to pursue family practice in sufficient numbers to meet the family practice manpower needs throughout the country.
- Support activities which enhance the educational preparation and training of prospective family physicians at the undergraduate and graduate levels.
- Support activities which maintain the educational excellence of family physicians throughout their practice experience.
- Support research activities in family practice which ultimately will result in improved patient care.

8. **What recent projects has FHFA been involved in?**

A. In 1981 and 1982, FHFA has awarded grants exceeding $600,000 in support of educational and scientific endeavors which promote and enhance family practice. These awards have been made to recipients in 13 different states as well as the AAFP, ABFP and Society of Teachers of Family Medicine. (Contact our executive vice-president Gary L. McMahan for details.) FHFA also plays an intricate and financial role in the Residency Development Project (ReD), The Residency Assistance Program (RAP), the Departmental Assistance Program (DAP), a project to develop a model curriculum outline for residency training called MERIT as well as administering the Mead-Johnson Awards. But that's only the beginning. . . .

9. **What about the new library?**

A. The Herb L. Huffington, M.D., Memorial Library is probably one of our most exciting projects yet. It was dedicated August 14, 1982. It includes history archives with taped oral interviews with distinguished family physicians, a research clearinghouse, journal and book collection, bibliographies of patient education data, with the extension of these basic reference services to Academy members via the WATS line.

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A. Service to the family practice movement, as a powerful ally. Certainly as family practice and family physicians encounter the many challenges the future holds for organized medicine and family practice, as well as periodic economic downturns, it is imperative that the family practice movement have a strong ally.

11. **Is a contribution to FHFA tax deductible?**

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Future Meetings

September

Metabolic Bone Disease and Topics in Orthopedic Nuclear Medicine, Kahler Hotel, Rochester, MN, Sept. 29-30. Fee: $75. 8.5 hrs. Category 1 credits. Contact: Deborah A. Churan, Exec. Dir., Cent. Chapt., SNM, Inc., 134 Lincoln Pkwy., Crystal Lake, IL 60014. Phone: (815) 459-6884.

October

Advanced Echocardiography, Hyatt Regency, Indianapolis, IN, Oct. 1-3. Fee: $405. 16.5 hrs. Category 1 credits. Contact: ACC, Extramural Prog. Dept., 9111 Old Georgetown Rd., Bethesda, MD 20814. Phone: (301) 897-5400.

American College of Chest Physicians' 50th Annual Scientific Assembly, Loews Anatole Hotel, Dallas, TX, Oct. 8-12. 30 hrs. Category I credits. Contact: Am. College of Chest Phy., P. O. Box 93826, Chicago, IL 60670. Phone: (312) 698-2200.

Clinical Aspects and Assessments of Alzheimer's Disease, Civic Center Inn, Eau Claire, WI, Oct. 12. Fee: $80. 0.4 hrs. CME credits. Contact: Cindy Marzofka, Prog. Cood., U. of Wisc., Scl. of Arts & Sciences, Outreach Off., Eau Claire, WI 54701. Phone: (715) 836-5179.

Principles of Colon and Rectal Surgery, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, Oct. 17-20. Fee: $375. 19.5 hrs. Category 1 credits. Contact: CME, U. of Minn., Box 293, 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Advanced Trauma Life Support, Orr Major Hall, U. of Kans. Med. Ctr., Kansas City, KS, Oct. 18-19. Fee: $350. 16 hrs. AMA Category I credits & 15 hrs. AAFP credits. Contact: Jody Scott, Dept. of Surg., U. of Kans. Med. Ctr., 39th & Rainbow, Kansas City, KS 66103. Phone: (913) 588-6124.

Conference on Medical Malpractice, Loews Summit Hotel, New York, NY, Oct. 18-20. Fee: $450. 18¾ hrs. Category 1 credits. Contact: Am. Coll. of Legal Med., 213 W. Institute Place, Chicago, IL 60610. Phone: (312) 440-0080.

Therapeutic Decisions in Contemporary Practice of Cardiology: Pharmacological Theory and Practical Applications, Kiawah Island, SC, Oct. 19-21. Fee: $355. 16.5 hrs. Category 1 credits. Contact: ACC, Extramural Prog. Dept., 9111 Old Georgetown Rd., Bethesda, MD 20814. Phone: (301) 897-5400.

Annual Autumn Seminar Obstetrics and Gynecology, Amfac Hotel, Minneapolis, MN, Oct. 24-26. Fee: $295. 17.5 hrs. AAFP & AMA Category 1 credits. Contact: CME, U. of Minn., Box 293 Mayo, 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Cardiology Conference and Third Annual Jesse E. Edwards Lectureship, Landmark Center, St. Paul, MN, Oct. 26-27. Fee: $75. 9 hrs. Category 1 credits. Contact: Medical Staff Office, United Hosps., 333 N. Smith Ave., St. Paul, MN 55102. Phone: (612) 298-8558.

52nd Annual Postgraduate Assembly, Red Lion Inn, Omaha, NE, Oct. 29-31. AAFP & AMA CME credits available. Contact: Omaha Midwest Clinical Society, 7363 Pacific St., Omaha, NE 68114. Phone: (402) 397-1443.

Obstacles and Solutions to Coping with Cancer, Menninger Found. Seeley Conf. Ctr., Topeka, KS, Oct. 29-30. Fee: $135. 12 hrs. Category 1 credits. Contact: Ms. Brenda Vink, Div. of CME, The Menninger Foundation, Box 829, Topeka, KS 66601. Phone: (913) 273-7500 ext. 5992.

November

Primary Care of the Child with a Development Disorder, Radisson Plaza Hotel, St. Paul, MN, Nov. 2-3. CME credits available. Contact: R. Brett Day, Ed.S., CME Dept., Gillette Children's Hosp., 200 E. Univ. Ave., St. Paul, MN 55101. Phone: (612) 291-2848 ext. 120.

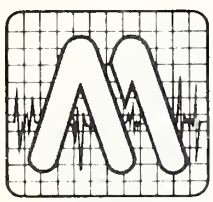
Pulmonary Medicine and Office Spirometry for the Primary Care Physician, Vacation Village Resort, San Diego, CA, Nov. 16-18. Fee: $295. 19 hrs. AAFP & AMA Category 1 credits. Contact: Off. of CME, UC San Diego School of Med., M-017. La Jolla, CA 92093. Phone: (619) 452-3940.

A Special Workshop on the Use of the CO_2 Laser in ENT Surgery, Sudler Hall, U. of Kans. Med. Ctr., Kansas City, KS, Nov. 29-30. Fee: $600. 16 hrs. Category 1 credits & 15 hrs. AAFP credits. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

Fourth Assembly Meeting of the AMA-Hospital Medical Staff Section (AMA-HMSS), Hilton Hawaiian Village, Honolulu, HI, Nov. 29-Dec. 3. Contact: AMA, Dept. of Hosp. Med. Staff Serv., 535 N. Dearborn St., Chicago, IL 60610. Phone: (312) 645-4747.

December

Ear, Nose and Throat Disease in Children: A 1984 Update, The Breakers, Palm Beach, FL, Dec. 5-9. Fee: $250. 17 hrs. CME credits. Contact: Dept. of Otolaryngology, Children's Hosp. of Pitts., 125 DeSoto St., Pittsburgh, PA 15213. Phone: (412) 647-5466.



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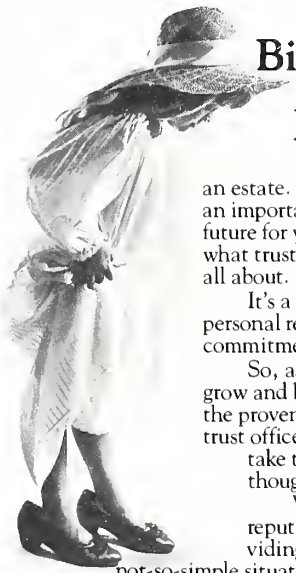

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Incidence less than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests; **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma; **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia; **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS); **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit; **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations; **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis; bronchospasm (see CONTRAINDICATIONS); **Renal:** Acute renal failure in patients with pre-existing significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria; **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence less than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis; **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri; **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions; **Special Senses:** Conjunctivitis, diplopia, optic neuritis; **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction; **Cardiovascular:** Arrhythmias (sinus tachycardia, sinus bradycardia); **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis; **Renal:** Renal papillary necrosis.

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SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 10 October 1984 (ISSN 0038-3317)

Official Publication of the
South Dakota State
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the South Dakota Chapter
of The American Academy
of Family Physicians

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Forty Year Old Man in Coma With Respiratory Distress After Falling Into a Hog Pit

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A Case of Unusual Theophylline Elimination in an Infant

David W. Ohrt, M.D., Ph.D.*
John S. Liggett, Jr., M.D.†

ABSTRACT

In the typical patient, theophylline clearance is a first order log linear process which allows linear dose adjustments. Over the past 10 years, several authors have alluded to occasional patients who have assumed a dose dependent, zero order elimination pattern similar to that seen with phenytoin. This is a report of an infant who demonstrated atypical responses to theophylline therapy which can be explained on the basis of intervening dose dependent elimination. Because of potential serious toxic manifestations at supratherapeutic levels, the assumption of zero order elimination kinetics is an important consideration when adjusting theophylline dosage.

Theophylline is a commonly administered drug for the treatment of asthma and neonatal apnea and has also been used with questionable efficacy in the treatment of Cheyne-Stokes respiration and congestive heart failure. The major physiologic effects of theophylline include central nervous system stimulation, smooth muscle relaxation, and diuresis. The drug is metabolized principally in the liver via a number of different degradative pathways with only about 10% being excreted directly by the kidneys in the typical patient. Since theophylline has a low therapeutic index, it is important to monitor plasma levels. Levels below the accepted therapeutic range of 10-20 mg/L characterizes the non-responders, whereas levels exceeding 25 mg/L are highly associated with toxic manifestations which include nausea, vomiting, agitation, arrhythmias, and seizures.

In earlier years of theophylline monitoring, the preponderance of attention was paid to the adequacy of the assay, but today sufficiently reliable, rapid, and economical methodology is available. With the resultant increase in utilization of theophylline assays, the occurrence of deviant pharmacokinetic behavior has become more widely recognized. In a typical patient, the theophylline level obtained at steady state may be used to directly transform the old dose into a new dose based upon a desired serum level as shown below.

$$\text{New Dose} = \frac{\text{Desired Serum Level}}{\text{Measured Serum Level}} \cdot \text{Old Dose}$$

Gradually increasing or decreasing the calculated new dose for subtherapeutic and supratherapeutic levels respectively constitutes the practical utilization of this mathematical expression.[1, 2] This equation can be applied when theophylline is eliminated by a first order kinetic process which is the usual case when plasma concentrations approximate the therapeutic range. If the kinetics of elimination should assume a dose dependent pattern, major problems in control may ensue. Dose dependent kinetics, also called zero order, Michaelis-Menton, or saturation kinetics refers to disproportionate increases in plasma level with dose increases. With first order kinetics a plot of steady state serum level vs. dose is linear indicating that drug clearance or

* Laboratory of Clinical Medicine, Sioux Valley Hospital; Consulting Pathologist, St. Mary's Hospital, Pierre, South Dakota; Clinical Assistant Professor, USD School of Medicine, Dept. of Laboratory Medicine, Sioux Falls, SD.
† Clinical Assistant Professor, USD School of Medicine, Department of Pediatrics; Director, Sioux Falls Cystic Fibrosis Center, Sioux Falls, SD, at the time the article was written. Now at U. of Texas Health Center, Tyler, TX.

halflife is independent of dose. If dose dependent kinetics is operative, the halflife increases as the dose increases resulting in a curvilinear relationship between the two entities as shown in Figure 1 below.[3, 4]

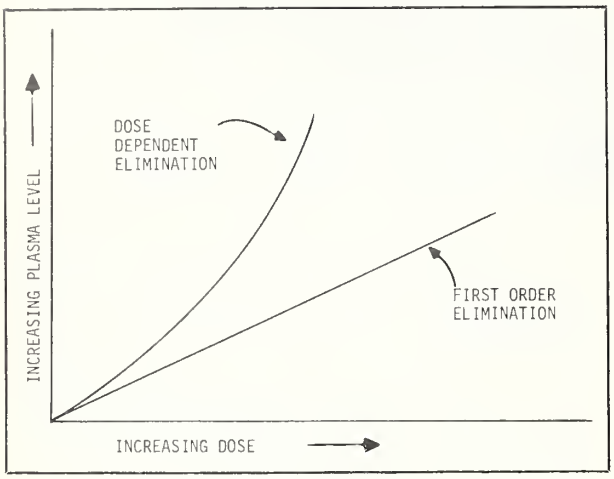

Figure 1
Relationship between plasma level and dose for first and zero order elimination kinetics.

We wish to report a case of unusual theophylline elimination in an infant and suggest an explanation based upon clinical correlation and analysis of the theophylline drug monitoring data obtained.

Case Presentation

The patient, presently a 1½-year-old white male, was delivered vaginally from a 34 year old G_4 P_3 AB_0 insulin dependent white gestational diabetic. The delivery was complicated by a large for gestational age infant weighing 4320 gm, and by gross meconium staining of the amniotic fluid with suspected aspiration. Immediately after delivery, coarse rales were heard over the right lung field which cleared after several deep breaths. An Apgar score of 4/7 was determined, and the head and chest circumferences were 37 and 38 cm respectively. The infant was admitted to the intensive care nursery with a PO_2 of 70 torr at 50% oxygen by hood. A chest film 1 day post delivery revealed evidence of meconium aspiration involving the right upper and mid lung fields. Oxygen requirements continued to decrease over the next four days with 23% oxygen being utilized by the fifth day. In spite of this, episodic problems with tachypnea, sternal retractions, and perioral cyanosis continued to occur. On the thirteenth hospital day, the infant was briefly intubated to remove bronchial mucous plugs from the right upper and right lower lobes. The apparent atelectatic processes in these regions did improve with some residual collapse in the right upper lobe. After twenty-two days in the hospital, the infant was discharged on vitamins with instructions for chest physiotherapy.

First Admission

The infant was readmitted four weeks after discharge with a two week history of respiratory difficulties that did not respond to bronchodilators and antibiotics. On the morning of admission, he developed severe respiratory distress which responded to subcutaneous epinephrine. Physical examination of the chest revealed some wheezing and atelectasis of the right lower lobe with a left perihilar infiltrate apparent on the chest film. There was a family history of apparent reactive airway disease. Admission blood gases were PO_2 — 40 torr, PCO_2 — 38 torr, pH — 7.38, CO_2 content — 23 meq/L, and 74% saturation. Complete blood count, electrolytes, and urinalysis were normal. A barium esophagram, upper gastrointestinal series, and sweat test for cystic fibrosis were within normal limits. The infant received a regimen of oral and inhaled sympathicomimetic agents, oral mucolytic agents, theophylline and corticosteroids. At a dosage of 90 mg/day theophylline in the form of oral Somophyllin Rx liquid, levels between 12-19 mg/L were obtained correlating with improvement in his respiratory status. His diagnoses included post-meconium aspiration, reactive airway disease, and chronic lobar collapse. After 24 days in the hospital he was discharged on Somophyllin Liquid Rx 1.3 cc po q 6 hours, and Metaprel Rx 1.5 cc po q 6 hours. Chest physiotherapy was to be performed 2-3 times per day.

Second Admission

At the age of three months, and 10 days after discharge, the infant was readmitted with intermittent cyanosis, dyspnea, and wheezing which failed to clear with medication. According to his mother, the patient had been receiving his medication and a history of vomiting or diarrhea could not be elicited. Physical examination revealed substernal retraction, mild tachypnea, tachycardia and auscultation of harsh inspiratory noise, and expiratory wheezes. Blood gases demonstrated a pH — 7.35, PCO_2 — 42 torr, CO_2 content — 22, PO_2 — 56 and an O_2 saturation of 84 percent.

An infiltrate in the right upper lobe consistent with atelectasis was demonstrated on the chest film. A theophylline level of 1.7 mg/L was obtained at the time of admission, and throughout his hospital course there were marked variations in theophylline levels with dosages varying from 90-209 mg/day. Therapy also included oral and inhaled sympathicomimetic agents, mucolytic agents, and prednisone. Two episodes of respiratory decompensation occurred which responded favorably to subcutaneous

epinephrine and nebulized atropine. After 30 days in the hospital, the infant was discharged to the Mayo Clinic for further workup including a lung biopsy which revealed changes compatible with reactive airway disease. His theophylline regimen was changed to Theodur Sprinkle Rx 75 mg q 6 hours.

Third Admission

The child was again admitted at the age of 7 months with respiratory difficulties associated with an upper respiratory tract infection. He would not take his feedings and there had been a few loose stools over the past several days prior to admission. Physical examination revealed a noncyanotic, obese child with a labored breathing that responded to 0.075 cc of subcutaneous epinephrine. The blood pressure, temperature, pulse and respirations were 97/66, 99°F, 180/minute, and 50/minute respectively. Scattered wheezes were heard over all lung fields and the chest film revealed lung hyperexpansion. During the first two days after admission, febrile episodes of up to 101°F occurred and an elevated theophylline level of 37.5 mg/L was assayed at this time. With the possible exception of some agitation, adverse symptoms were not identified. Over the course of the admission, widely fluctuant theophylline levels were obtained. After six days of therapy, the patient was discharged on Theo-Dur Sprinkle Rx, corticosteroids, Metaprel Rx, Bronchosol Rx, and cromolyn sodium.

Fourth Admission

The child was again admitted at the age of 10 months with increased cough, wheezing, and sternal retractions; a clinical setting quite similar to the previous admissions. The chest film again revealed a right lung infiltrate.Arterial blood gases demonstrated a pH — 7.41, pCO_2 — 36 torr, a CO_2 content — 24 meq/l, and pO_2 — 55 torr with 80% saturation. There was a mild leukocytosis of 13.8 x 10^9/L with 78% segs, 3% bands, 16% lymphocytes, and 3% monocytes noted on CBC. A markedly elevated serum theophylline level of 39.9 mg/L occurred during a febrile episode on the second day and was associated with symptoms of mild agitation. Throughout his hospitalization, much difficulty with theophylline control was again encountered. A few days prior to discharge his respiratory status improved markedly and after 15 days he was discharged on his usual regimen.

Fifth Admission

At the age of 11 months, he was admitted with a fever of 101°F and a history of vomiting. There were bilateral coarse breath sounds on physical examination and the chest film did reveal haziness in the right perihilar area. Blood gases demonstrated a pH and pCO_2 of 7.41 and 31 torr respectively and the electrolytes were within normal limits. The white blood count was 21.4 x 10^9/L with 58 segmented neutrophils, 23 band neutrophils, and 19 lymphocytes with increased platelets. Numerous highly variant theophylline levels were assayed and an extremely high level of 45 mg/L occurred during a febrile episode. He was stable after 8 days and discharged.

Materials and Methods

During the course of this patient's many admissions for reactive airway disease numerous theophylline levels were obtained to monitor therapy. When on oral theophylline therapy, the peak levels for Somophyllin Elixer Rx and Theo-Dur Sprinkle Rx were drawn between 1-2 hours and 3-5 hours after the dose respectively. All trough levels were drawn within 30 minutes of the next dose. On the few occasions when intermittent intravenous therapy was used, peak theophylline levels were drawn approximately 30 minutes after a 30 minute infusion. Upon collection by heelstick, the specimens were allowed to clot and the serum removed and refrigerated prior to assay by the SYVA EMIT technique using a Multistat III Microcentrifugal Analyzer.[5] With each theophylline assay, three levels of control sera were assayed during the same run, and review of the quality control charts did not demonstrate variances beyond the accepted two standard deviations from the mean value for each control. Coefficients of variation ranged from 3 to 6 percent with the best precision being noted towards the lower end of the therapeutic range.

Results

Since aberrant theophylline levels were not noted during the course of the first of the five admissions, only the drug monitoring data for the last four admissions is tabulated in Tables I-IV. The last three admissions were characterized by theophylline levels well into the toxic range which occurred during febrile episodes in all instances.

In Table V, the theophylline clearance ranges are summarized. The steady state clearance calculations for continuous IV therapy utilized the following basic equation:

Clearances for intermittent IV and oral therapy were obtained in a similar fashion by first calculating a simulated infusion rate by dividing the daily dose by 24 hours. For sake of consistency in regards to intermittent therapy, only theophylline levels at presumed steady state were used for the estimation of clearance. The calculated clearance must be viewed as an estimate since it assumes complete absorption of drug, a steady state serum level, as well as a constant fixed volume of distribution.

TABLE I
THEOPHYLLINE MONITORING DATA: SECOND ADMISSION

DATE	DOSE THEOPHYLLINE (Mg/Day)	FORMULATION	MODE OF ADMINISTRATION	LEVEL (Mg/L)
Adm.	90	Somophyllin	Oral	1.7
6-9	90	"	"	5.3 — P†
6-9	90	"	"	2.8 — T†
6-13	117	"	"	8.1 — P
6-13	117	"	"	4.2 — T
6-14	130	"	"	16.1 — P
6-18	130	"	"	11.8 — P
6-19	135	Aminophyllin	Int. IV	14.4 — P
6-21	136	"	"	19.0 — P
6-25	143	"	Cont. IV	8.6
6-26	163	"	"	6.6
6-28	173	"	"	12.6
6-29	179	"	"	11.2
6-29	179	"	"	9.9
6-30	204	"	"	15.2
7-1	204	"	"	23.2
7-1	204	"	"	23.0
7-2	194	"	"	18.2
7-3	194	"	"	17.0
7-4	204	"	"	13.4
7-5	204	"	"	11.4
7-6	209	"	"	11.3

Transferred to MAYO Clinic on 7-6-82

TABLE II
THEOPHYLLINE MONITORING DATA: THIRD ADMISSION

DATE	DOSE THEOPHYLLINE (Mg/Day)	FORMULATION	MODE OF ADMINISTRATION	LEVEL (Mg/L)
10-10	300	Theo-Dur Sprinkle	Oral	18.0
10-12	306	Aminophyllin	Cont. IV	37.5*
10-12	D/C	D/C	D/C	20.0
10-13	300	Theo-Dur Sprinkle	Oral	9.8
10-14	300	"	"	17.1

Discharged 10-16-82 to Home

TABLE III
THEOPHYLLINE MONITORING DATA: FOURTH ADMISSION

DATE	DOSE THEOPHYLLINE (Mg/Day)	FORMULATION	MODE OF ADMINISTRATION	LEVEL (Mg/L)
Adm.	300	Theo-Dur Sprinkle	Oral	26.1
1-3	326	Aminophyllin	Cont. IV	39.9*
1-3	102	"	"	31.1
1-4	102	"	"	11.9
1-5	143	"	"	11.7
1-5	143	"	"	11.5
1-6	184	"	"	11.8
1-7	245	"	"	15.5
1-9	300	Theo-Dur Sprinkle	Oral	6.9
1-10	300	"	"	12.2
1-11	300	"	"	14.2 — T†
1-11	300	"	"	15.9 — P†
1-13	300	"	"	36.0 — P
1-13	300	"	"	32.4 — T
1-13	225	"	"	27.7 — T

Discharged Jan. 15, 1983

TABLE IV
THEOPHYLLINE MONITORING DATA: FIFTH ADMISSION

DATE	DOSE THEOPHYLLINE (Mg/Day)	FORMULATION	MODE OF ADMINISTRATION	LEVEL (Mg/L)
Adm.	225	Theo-Dur Sprinkle	Oral	19.4 — T
2-4	240	Aminophyllin	Int. IV	45.0 — P*
2-5	Dose held	”	”	22.0 — T†
2-5	184	”	”	13.2 — P†
2-6	184	”	”	13.0 — P
2-7	190	”	”	19.8 — P
2-10	225	Theo-Dur Sprinkle	Oral	15.8 — P
Discharged 2-11-83				

*Patient febrile
†T and P refer to peak and trough respectively

TABLE V
CALCULATED CLEARANCE RANGES FOR LAST FOUR ADMISSION

ADMISSION	WEIGHT (Kg)	CLEARANCE RANGE (Mg/Hr)
6-6-82	6.2	0.4-1.3
10-10-82	7.5	0.3-0.7
1-2-83	8.2	0.3-0.9
2-3-83	8.5	0.2-0.6

$$\text{Clearance (L/Hr)} = \frac{\text{Theophylline Infusion rate (Mg/Hr)}}{\text{Steady State Serum Concentration (Mg/L)}}$$

The bulk of the representative information is contained in Tables I and III. Figure 2 illustrates the relationship between steady state serum level and dose using the data from Table I. Only trough levels or continuous IV theophylline levels at presumed steady state were used in preparing this plot. The uniform selection of trough levels provides consistency for comparative analysis and trough levels tend to be regarded by many as the standard for routine theophylline monitoring. The data incurred from the time of admission to 7-7-83 is used in the plot. Interestingly the levels began to decrease after 7-7-83, correlating with an improvement in the patient's respiratory status. The curvilinear nature of the plot is evidence for the occurrence of dose dependent kinetics over a significant portion of the hospital admission.

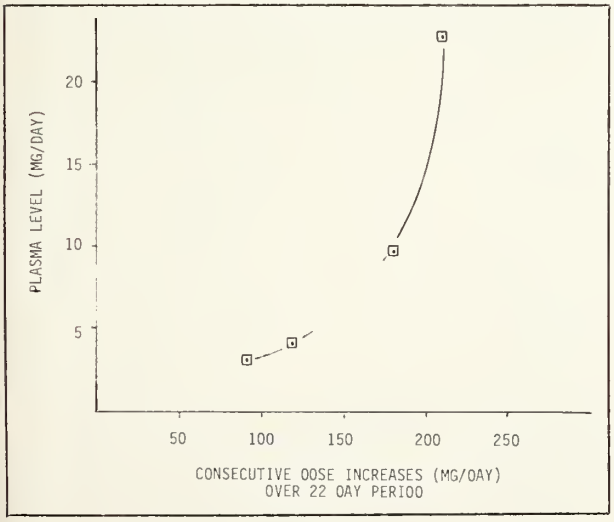

Figure 2
Serum level of theophylline vs dose at steady state for second admission.

Figure 3 is a plot of steady state serum level vs. dose using the data contained in Table III. The steady state levels obtained between 1-4-83 and 1-13-83 are illustrated in the plot. The levels obtained during the febrile episode are not included since there is uncertainty regarding the presence or absence of steady state.

The first part of the curve is flat illustrating a lack of response despite increases in dose. An increase in serum level did not occur until a daily dose of 245 mg was reached, and at 300 mg/day a marked increase in plasma level resulted and suggests a transition to dose dependent elimination.

Figure 3 provides an explanation for the lack of response to dose increases. At the time of the febrile episode, the clearance was estimated to be less than 0.4 L/Hr. The clearance increased following the fever and continued to increase until a dosage of 245 mg/day was reached. The apparent clearance then began to decrease with dose increases in accordance with saturation kinetics.

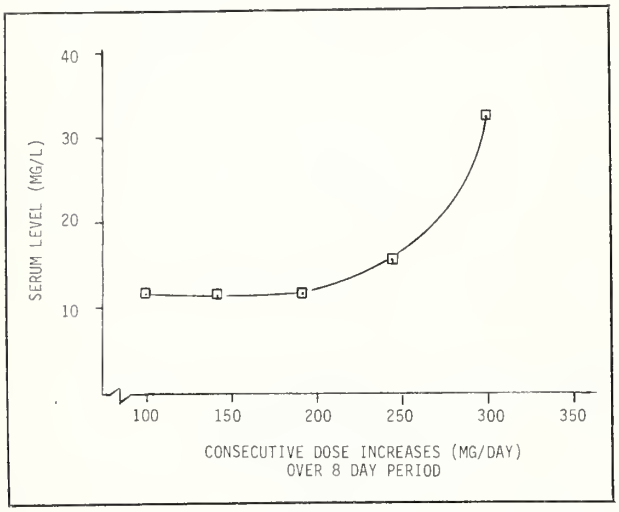

Figure 3
Serum theophylline levels vs dose at steady state on fourth admission.

Discussion

The elimination mechanism for theophylline has generally been considered to be a log linear first order process. This indicates that the rate of elimination is independent of the theophylline dose administered and that clearance measurements and halflife determinations will not vary with the dose. Over the past 5-10 years, this concept has been challenged. Students of pharmacokinetics have become increasingly aware that the metabolism of theophylline is not a well understood phenomenon. In 1972, Jenne reported that serum theophylline concentrations tended to increase or decrease disproportionately with the dose in an occasional patient. In one instance the serum level increased by a factor of three with a twofold increase in dose.[7] In 1977, Weinberger and Ginchansky published a systematic study of theophylline levels in asthmatic children and demonstrated disproportionate changes in serum levels with dose.[8] Kadlec et al reported a case of acute theophylline intoxication in a child. Theophylline levels in this case were followed from 100 mg/L to approximately 5 mg/L with two patterns of elimination being noted over this concentration span. One pattern was characterized by a halflife of 12.6 hours occurring over a serum theophylline concentration range of 100-20 mg/L and another having a halflife of 5.4 hours which occurred from 20 to 5 mg/L. Review articles on theophylline disposition in patients by Ogilvie and Lesko also attest to the need for systematic studies of theophylline elimination as well as the probable occurrence of zero order elimination.[4, 9]

Theophylline elimination is complex due to the presence of multiple degradative pathways in the liver. Figure 5 illustrates the three known pathways of elimination which include demethylation and oxidative mechanisms.

In 1976, Jenne studied the urinary theophylline metabolite excretion in patients after chronic dosing to determine if metabolite excretion was a function of plasma level and found the major pathway of excretion to be 1-demethylation which is responsible for the production of 3-methylxanthine. As the plasma concentration increased the fraction of 3-methylxanthine in the urine decreased suggesting that this pathway is saturable. At higher concentrations oxidation to 1,3-dimethyluric acid became more prominent and appeared to be linearly related to plasma theophylline concentrations throughout this entire range. This data suggested that Michaelis-Menton kinetics may govern the 1-demethylation pathway which accounts for approximately 36% of theophylline metabolism.[10]

The patient under discussion demonstrates an elimination pattern similar to those described in the literature. As indicated in Figure 2 and Table I, a change in dose from 179 to 204 mg/day resulted in a twofold increase in theophylline level. The overall appearance of the curve would be consistent with dose dependency. The interpretation is further complicated by the effect of disease on elimination which must be addressed since the plasma level dropped from 23 to 11 mg/L over a 3 day period as shown in Table I and seemed to correlate with a much improved clinical status. The effect of disease is also a factor in the interpretation of Figure 4 in view of the effect of fever on clearance. After the febrile episode subsided, the apparent clearance progressively increased as the dose was increased providing an explanation for the failure of the plasma level to increase in a predictable fashion as shown in Figure 3. At higher doses, however, disproportionate increases in the plasma levels due to decreased clearance were observed without an apparent clinical correlate since the patient was doing well over this time interval. It is impossible in this case to clearly distinguish between pure dose dependency and the intermittent effects of disease. Upon relief of severe obstruction during the second admission the clearance increased suggesting that disease may be instrumental in the pharmacokinetic transformation observed. The numerous affectors of theophylline elimination are well recognized and

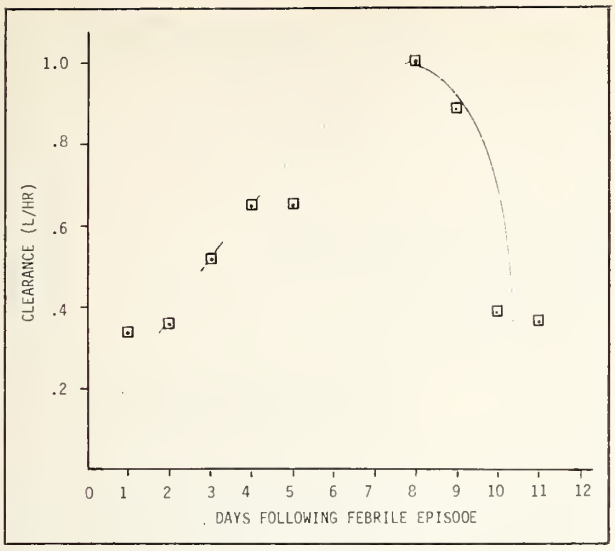

Figure 4
Clearance as a function of days following febrile episode; fourth admission.

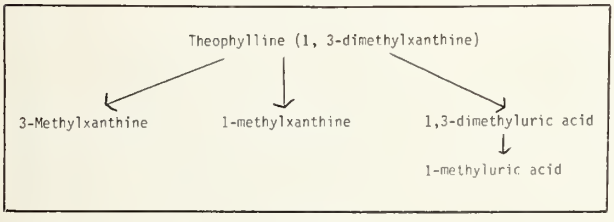

Figure 5
Proposed metabolic pathway for theophylline elimination.

include age, liver disease, congestive heart failure, smoking, obesity, chronic lung disease, fever, upper respiratory tract infections, pneumonia, macrolide antibiotics, and quantity of dietary carbohydrate. Of the affectors indicated premature infancy, congestive heart failure, and liver disease are known to markedly prolong the halflife of elimination. The influence of disease upon the kinetic mode of theophylline elimination is not well described and may well be partially responsible for the apparent Michaelis-Menton kinetics observed in this case. It would be interesting to investigate whether dose dependent or first order elimination kinetics is operative in those patients having diseases or circumstances that are known to decrease theophylline elimination.

The presence or absence of dose dependency has considerable practical significance in the use of theophylline. Indeed the data obtained in this case suggests that disease may induce a transformation from first order to dose dependent elimination. There is also evidence to suggest that a purely dose dependent process may be operative in the absence of overt disease. In the case at hand severe respiratory obstruction, upper respiratory tract infections, and febrile episodes were present and probably instrumental in producing the unusual response to the theophylline regimens. If the presence of dose dependency could be predicted, more effective dose adjustments could be initiated, especially in the acute care setting.

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The ever-changing role of federal and some times state government in the delivery of health care services places a serious strain on the physician-patient relationship. With the advent of new programs and regulations that infringe on private medicine the principles of medical ethics need current reaffirmation. Dr. Joseph Boyle in his inaugural address asked us to make this one of our prime projects during the coming year. The 1984 issue of the AMA Judicial Council elucidates these principles very well. Historically ethical principals for medical practice date from about 2000 B.C. to the present. The Oath of Hippocrates was conceived in about the fifth century B.C. protecting the rights of the patient and appealing to the finer instincts of the physician. In 1847, the first official meeting of the AMA of Philadelphia established a code of ethics. Since then changes have been made periodically. The most recent revision was adopted in 1980, as follows:

I. A physician shall be dedicated to providing competent medical service with compassion and respect for human dignity.
II. A physician shall deal honestly with patients and colleagues, and strive to expose those physicians deficient in character or competence, or who engage in fraud or deception.
III. A physician shall respect the law and also recognize a responsibility to seek changes in those requirements which are contrary to the best interests of the patient.
IV. A physician shall respect the rights of patients, of colleagues, and of other health professionals, and shall safeguard patient confidences within the constraints of the law.
V. A physician shall continue to study, apply and advance scientific knowledge, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated.
VI. A physician shall, in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve, with whom to associate, and the environment in which to provide medical services.
VII. A physician shall recongize a responsibility to participate in activities contributing to an improved community.

Utilizing these principles of medical ethics, we can continue to provide the worlds best medical care, available to all who need care, in an appropriate manner, at a reasonable cost. While this all sounds idealistic, I'm sure if we all put our shoulder to the wheel we can accomplish the goal.

Howard L. Saylor Jr.

Howard L. Saylor, Jr., M.D., President
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Thirteen Month Old Caucasian Male Admitted With Extreme Pallor

Debra G. Peterson, M.D.*
Bert W. Larson, M.D.†
Discussers

John F. Barlow, M.D.‡
Editor

Case #1-030-429

This 13-month old previously healthy Caucasian male was admitted to Sioux Valley Hospital because of extreme pallor.

The patient had been well until approximately three weeks prior to admission when he developed nonbloody diarrhea. He had been exposed to other children with these symptoms. Stool examination revealed Giardia lamblia cysts. The patient was treated with 6cc of liquid furazolidone four times a day for eight days. The diarrhea abated until 1½ weeks prior to admission but the recrudescence responded to oral electrolyte diet. Three to four days prior to admission the patient developed red or "iodine" colored stools and two days before admission had red or dark urine. One day prior to admission the patient developed puffiness of the lips and periobital regions rapidly spreading to the hands and feet. The patient was brought to the emergency room where he was noted to be extremely pale. Hemoglobin was 6.5 gm/dl. There was no history of chills, fever, sweats, but the patient had had some mild rhinorrhea and nonproductive cough without hemoptysis for the last three or four days. There had been a vomiting epoisode without hematemesis. There was no history of previous hospitalizations or other medications. Immunizations were up to date and the obstetrical history revealed that the patient did aspirate some fluid at birth with cyanosis and was in the intensive care unit for nine days. The patient seemed to recover from this episode and had progressed normally until about two months ago when the growth curve showed a decline in growth rate.

Physical examination revealed a severely distressed, pale infant with nonpitting edema of the hands and feet. Temperature was 37.5°C, pulse 124/minute and regular, respirations 34/minute and regular, blood pressure 109 systolic and 77 diastolic. Weight 10 kg. Examination of the head and neck was unremarkable. The ear canals were blocked by cerumen. There was no palpable lymphadenopathy. The chest was clear to auscultation and percussion. There was tachycardia and a grade I apical systolic murmur. The pulses were equal and unremarkable in the hands and feet. Examination of the abdomen was difficult because the patient was crying. The presence of bowel sounds could not be assessed but there seemed to be no palpable organs or masses. Neurologic examination appeared intact.

LABORATORY DATA: Urinalysis — brown, hazy, specific gravity 1.016, pH 7.0, 3+ protein and 3+ hemoglobin, negative for glucose, reducing substances, ketones and bilirubin; sediment was not done on the initial urinalysis due to insufficient quantity but subsequent urinalysis showed 100 to 150 rbc/hpf, 15-20 wbc/hpf, and 1-2 hyaline casts/lpf. Total urinary protein excretion was 150 mg for 12 hours which is slightly elevated; hemogram — hemoglobin 6.5 gm/dl, hematocrit 20%, red count 2.4 × 10^{12}/L, mean corpuscular volume 80 FL, mean corpuscular hemoglobin 27 PG, mean corpuscular hemoglobin concentration 33%, total leukocyte count 12.5 × 10^{9}/L with a differential count of 29% segmented neutrophils, 3% neutrophilic bands, 62% lymphocytes with an occasional reactive form and 6% monocytes; platelet count 72 × 10^{9}/L; erythrocyte sedimentation test 19 mm/hr; reticulocyte count 2% on admission rising to 14% by the third hospital day. The peripheral blood smear showed marked anisocytosis and poikilocytosis of the red cells. There were scattered spherocytes and moderate numbers of schistocytes. There was mild polychromasia.

A direct antiglobulin test was negative. A test for fecal pH and reducing substances was negative. A prothrombin time and partial thromboplastin time were within normal limits. A fibrinogen, total bilirubin and glucose were within normal limits; blood urea nitrogen 68 mg/dl, creatinine 1.0 mg/dl; creatinine clearance was 25.2 cc/minute (normal 80-135 cc/minute); calcium 8.3 mg/dl (normal 8.9 to 11.3 mg/dl), phosphorus 5.2 mg/dl (normal 2.0 to 4.6 mg/dl); serum osmolality 311 mos/kg (normal 280 to 300 mos/kg); urine osmolality normal; transaminase (SGOT, AST) 47 IU/L (normal 0-30 IU/L); venous pH 7.34; pCO2 35 torr; sodium 144 meq/L; potassium 5.4 meq/L; chloride 113 meq/L; and CO2 content 22 mm/L; total protein 4.5 gm/L (normal 6.0 to 7.7 gm/L) with albumin 2.8 gm/L, alpha I globulin 0.4 gm/L, alpha II globulin 0.5 gm/L, beta globulin 0.5 gm/L and gamma globulin 0.3 gm/L. (The gamma globulin was slightly decreased.) Tests for glucose 6 phosphate dehydrogenase

* Resident in Family and Community Medicine, affiliated with USD School of Medicine, Sioux Falls, SD.
† Resident in Pathology and Laboratory medicine, Sioux Valley and VA Hospitals, affiliated with USD School of Medicine, Sioux Falls, SD.
‡ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital. Professor of Pathology, USD School of Medicine, Sioux Falls, SD

and complement fractions C4 and C3 were within normal limits. Haptoglobin was markedly reduced. Three stools for occult blood were negative. There were no ova and parasites and only a rare leukocyte was seen in the stool. A urine culture showed no growth after 48 hours. A stool culture revealed no pathogens. Blood culture showed one bottle growing Streptococcus pneumoniae sensitive to all the antibiotics tested. A chest film revealed clear lung fields with a slightly prominent cardiac silhouette. A supine and upright abdomen revealed no evidence of abdominal disease.

DR. PETERSON: I would like to know before I start whether this patient was oliguric?
DR. BARLOW: According to the chart the patient was not significantly oliguric.
DR. PETERSON: This 13-month old child had three distinct disease processes:

1) hemolytic anemia
2) nephritis with hematuria and azotemia
3) thrombocytopenia

It might be wise to list some of the causes of these three disorders and discuss the differential diagnosis to see how they best fit.

Causes of Hemolytic Anemia

1. Renal disease
2. Hereditary spherocytosis
3. Thalassemia
4. Immunohemolytic disease
5. Parasites such as malaria
6. Abnormal hemoglobins such as sickle cell anemia or sickle C disease
7. Acute oxidant injury occurring in a patient with a red cell enzyme deficiency e.g. G6PD
8. Paroxysmal nocturnal hemoglobinuria
9. Infections eg. clostridial sepsis
10. Toxins or chemicals — copper, snake or spider venoms and infusions with the distilled water such as can occur in drowning in fresh water.

Causes of Thrombocytopenia

1. Idiopathic or immune thrombocytopenic purpura (ITP)
2. Thrombotic thrombocytopenia purpura (TTP)
3. Drug induced immune thrombocytopenia
4. Leukemias

Causes of Azotemia and Nephritis

1. Post streptococcal
2. Drugs
3. Hemolytic uremic syndrome (HUS)
4. Idiopathic nephritis

The patient had several pertinent findings including a history of treatment of Giardia lamblia with furazolidine, a drug which has been known to produce acute hemolysis in patients with red cell enzyme deficiency such as G6PD. The test for G6PD deficiency was negative in this case. The patient had diarrhea and symptoms of an upper respiratory infection prior to admission. The extreme pallor and grade I systolic murmur with tachycardia were probably secondary to the severe anemia.

There were several pertinent laboratory findings which included 3+ protein and hemoglobin in the urine with a urinary sediment showing 100 to 150 red blood cells per high field and 15 to 20 leukocytes per high power field and 1 to 2 hyaline casts per low power field. This certainly suggests renal damage and I wonder about a glomerulonephritis in spite of the fact that no red cell casts were noted. The peripheral blood smear was particularly striking with many spherocytes and schistocytes. This picture strongly suggests microangiopathic hemolytic anemia. A markedly reduced serum haptoglobin also suggests hemolysis.

The patient also had a significant elevation of blood urea nitrogen and creatinine and a severe thrombocytopenia. A possibly significant finding is the presence of Streptococcus pneumoniae in one bottle from a blood culture.

According to the differential diagnosis and pertinent findings as I have outlined them, I think one could come to only one conclusion — the patient had hemolytic uremic syndrome (HUS).

Hemolytic uremic syndrome is a triad associated with:

1) acute renal failure often with a mixed nephrotic and nephritic picture
2) microangiopathic hemolytic anemia
3) thrombocytopenia

There is often a close resemblance between HUS and thrombotic thrombocytopenic purpura (TTP), but patients with the latter disease often have more prominent neurologic findings secondary to fibrin thrombi in the microcirculation of the central nervous system. TTP occurs more frequently in adults, but HUS is more frequently seen in young children as in this case. The adults with TTP have a significantly worse prognosis than do children with HUS.

In children with HUS there is usually a preceding infection which is most often gastrointestinal. In fact, there have been recent cases of HUS following Campylobacter jejuni infection. However, upper respiratory tract infections can also precede HUS. This prodromal infection is followed within a few days by the rapid onset of pallor and oliguria. These patients are usually under the age of 2 (most commonly between 4 to 12 months of age). The disease occurs sporadically in temperate climates and has a very high incidence in warm areas of the world. Large numbers of cases have been reported from Los Angeles and Buenos Aires. Infections or phenomena triggering the disease have included shigellosis, typhoid fever, diphtheria, pertussis, tetanus im-

munization (DPT), and various viral infections including coxsakie, echo, influenzae and myxovirus infections.

The pathogenesis of the disease has not been determined but several factors have been linked to the etiology of the disease:

1) genetic factors may be important since the disease is seen in family members
2) prostacyclin deficiency
3) infections by organisms producing neuraminidase such as Streptococcus pneumoniae (pneumococcus) in this case
4) endotoxins from such agents as Shigella
5) antithrombin III deficiency
6) deficiency of antioxidants in red cells, e.g. G6PD

The anemia in the disease is typically microangiopathic and is characterized by a low serum haptoglobin, increased bilirubin and often an inappropriately low reticulocyte count which was seen early in this patient's disease. The red cells are apparently damaged by fibrin strands in the vessels of the kidney. Mechanical injury seems to be the main problem but this red cell fragmentation may be facilitated by a decrease in antioxidants. Damage to red cells are then often seen sequestered in the spleen, a phenomenon which can be demonstrated by demonstrating splenic sequestration after radioactively tagging the patient's red cells. Neuraminidase producing organisms such as pneumococcus may indeed cause red cell damage or agglutination with secondary splenic sequestration and anemia. The thrombocytopenia usually lasts up to 14 days and seems to be due to peripheral platelet destruction. Some studies have demonstrated platelet antibodies to be present several weeks after onset of the HUS.

The renal injury in HUS is characterized by:

1) cortical necrosis
2) predominant glomerular involvement with capillary fibrin thrombi
3) Arteriolar involvement (fibrin thrombi)

The glomerular injury is primarily to the capillary endothelial cell. This injury may be secondary to endotoxin, neuraminidase or estrogen containing oral contraceptive agents. The latter is only a factor in HUS seen in adult women. In addition to the tissue damage described, HUS can be associated with symptoms due to involvement of the gastrointestinal tract, liver, heart and central nervous system. 20% of patients may present with a picture of acute abdominal pain often with bloody diarrhea and abdominal tenderness.

The mortality rate of HUS, at one time, was as high as 30% but now has improved to 4 to 7% due to better management of the condition. Management includes:

1) transfusions when indicated
2) careful control of electrolytes and water balance
3) lowering the blood pressure in cases in which hypertension occurs
4) peritoneal dialysis when indicated

Studies investigating the role of heparin, streptokinase, dipyridamole, and aspirin in treatment of HUS have not shown conclusive benefits from these agents. There are several clinical indications of a poor prognosis and these include: a nongastrointestinal prodrome, familial involvement, HUS in pregnancy or adults, progressively declining renal function, persistent or recurrent thrombocytopenia and central nervous system involvement.

Dr. Peterson's Diagnosis: Hemolytic Uremic Syndrome

DR. BARLOW: The patient responded very well to conservative therapy with disappearance of the azotemia and rise in the platelet count to normal and elevation in the hemoglobin level. He was discharged afebrile on the tenth hospital day.

DR. LARSON: I would like to begin the discussion of the laboratory aspects of the case with a tour of the patient's peripheral smear since the hematologic findings are quite striking in this syndrome. At low magnification a shortage of platelets plus marked anisocytosis and poikilocytosis of red cells or variation in their size and shape is seen. The white blood cells appear normal in number in contrast to the marked leukocytosis often seen in this disorder.[1] At higher magnification we see once again the pronounced thrombocytopenia, which is present in 85% of cases. The platelet count will generally be less than 140×10^9/L upon admission and usually does not fall below 20×10^9/L.[2] If we were to examine the marrow of this patient, we would see megakaryocytes of normal number and morphology, suggesting peripheral consumption rather than deficient production as the basis for the reduced platelets. In vitro platelet function abnormalities have also been described in aggregation studies, apparently unrelated to the renal function abnormality.[3] The classic finding in the red cell series is the presence of schistocytes or schizocytes, i.e. fragmented red cells (Fig. 1) The depressive effect of these on the mean cell volume is offset by the prominent reticulocytosis unless another hematologic process such as iron deficiency supervenes.[4] We see the reticulocytes as enlarged cells with polychromasia and occasional basophilic stippling. Reticulocyte counts in hemolytic uremic syndrome are usually greater than 3% and range to about 15%,[4] consistent with that seen in the present case. An occasional spherocyte is seen as further evidence of ongoing hemolysis. Other cells,

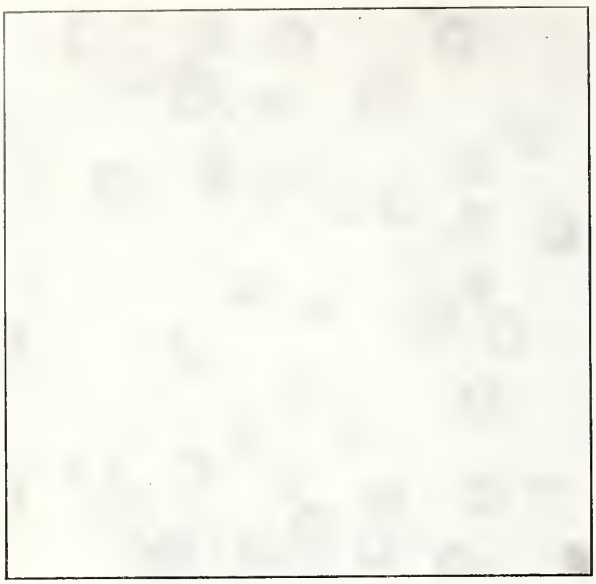

Figure 1

Peripheral blood smear showing several schistocytes (broken up irregular helmet shaped cells), three large "blue" polychrome cells indicating reticulocytes and low platelets. These findings are all typical of microangiopathic hemolytic anemia.

such as target cells and cells with a sickled appearance, may suggest a hemoglobinopathy. Confusion with sickle cell anemia is not typically a problem, however, since the hemolytic uremic syndrome is rare in blacks. A hemoglobin electrophoresis would easily resolve this issue. The source of red cell destruction in these patients has traditionally been ascribed to their passage over fibrin strands in the renal microvasculature. This no doubt occurs, yet other mechanisms of red cell damage have been invoked.[3]

Unfortunate from an academic view is the lack of a renal biopsy in this case. It is of interest that there are parts of the world where hemolytic uremic syndrome constitutes a substantial percentage of admissions for glomerular disease.[2] It is a major cause of acute renal failure in infants,[1] and moderate numbers of cases are also reported in adult patients.[1, 4] The renal changes may vary considerably from individual to individual and within a given specimen.[5] The main process appears to be endothelial disruption, and it may affect primarily the glomerular capillaries, the renal arterioles, or both.[3] Affected glomeruli may show only mild cellular proliferation, ranging to more prominent mesangial proliferation, focal crescent formation or segmental necrosis, and capillary collapse and sclerosis.[5] Fibrin thrombi are not required to make the diagnosis,[2] but may be present in abundance and associated with red blood cell fragments. Arterioles and small arteries may show thrombosis, and patients with this finding have a less favorable prognosis[3] and are likely to be hypertensive. Arteriolar aneurysms with thrombosis and organization, so-called glomeruloid structures, may be identified.[5] Extensive arteriolar damage may be seen with associated cortical necrosis, as was described in a Clinicopathologic Conference case in the New England Journal of Medicine several years ago.[4]

Electron microscopy may show separation of the swollen endothelial cells from the basement membrane with interposed debris in the resulting space. This material may contain fibrin and red cell or platelet fragments.[5] The basement membrane is usually intact and electron dense deposits are not identified, but the mesangium may be edematous and lytic, lacking homogeneity. There may also be extensive foot process fusion.[2]

Hemolytic uremic syndrome has been distinguished from thrombotic thrombocytopenic purpura by the relative prominence of the renal abnormality and by the corresponding lack of findings outside the renal and hematologic systems. As has been mentioned in the earlier discussion, however, these are not the only affected systems. Gastrointestinal manifestations may be severe, and the disorder may clinically and pathologically resemble ulcerative colitis. That the patient may present with an acute abdomen was pointed out by Dr. Peterson and also by a discussant at this conference five years ago, who described two children in this community who underwent appendectomies before the correct diagnosis of hemolytic uremic syndrome was made.[6] Although neurologic symptoms including seizures may be prominent, neuropathologic examination reveals only nonspecific ischemic or hypoxic changes, as opposed to the microangiopathic alterations in the central nervous system seen with thrombotic thrombocytopenic purpura.[2]

Of incidental interest in this case are the microbiologic data. Although pneumococcus is not a typical blood culture contaminant, its recovery from only a single culture bottle and the negative chest film dampen its significance. In addition, however, Giardia lamblia cysts were discovered in a stool sample during the prodromal period. Although this is a ubiquitous gastrointestinal pathogen and numerous other gastrointestinal pathogens, bacterial and viral, have been described in association with hemolytic uremic syndrome, I have not encountered reports of the syndrome in association with giardiasis.

Finally, of epidemiologic interest is an article in the November 11, 1983 Morbidity and Mortality Weekly Report[7] describing 32 cases occurring between June and October in 13 centers on the East Coast. These children had a bloody or, less commonly, a non-bloody diarrheal prodrome; 2/3 re-

quired dialysis, and 2 patients have persistent renal disease and neurologic sequelae. The hemolytic uremic syndrome is, thus, a disorder of some currency, and the Centers for Disease Control are interested in hearing of additional cases.

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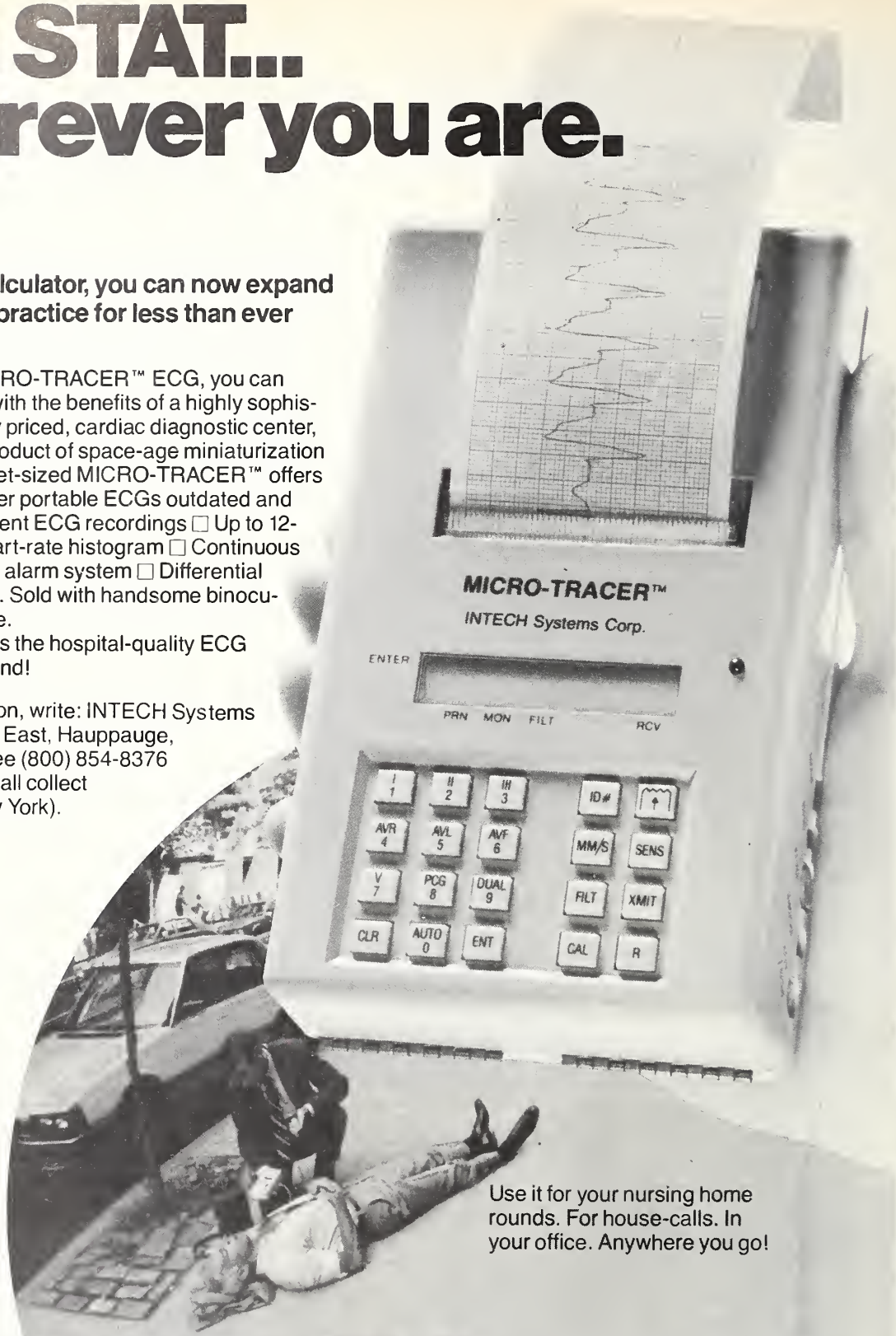

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   2. Nominee must be and have been actively engaged as a family physician in South Dakota, a majority of which time must have been in active practice.
   3. Nominee must be certified and hold a current recertification certificate from The American Board of Family Practice.
   4. Nominee must be of good moral quality and standing, and hold an unrestricted license to practice medicine in South Dakota.
   5. Nominee must be an active member of at least one hospital medical staff.
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      b. A current curriculum vitae.
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II. Selection process:
   1. Award developed and sponsored by The South Dakota Academy of Family Physicians.
   2. This is to be an annual Award, however, Award need not be given annually, at the discretion of the Selection Committee.
   3. Selection process developed and conducted by The South Dakota Academy of Family Physicians.
      a. Nominations may be made through any SDAFP member.
      b. Deadline for annual nominations is December 15 of the Year prior to which the Award is to be made.
      c. The SDAFP Public Relations Committee, composed of no less than 3 members, shall act as a Screening Committee.
      d. The Screening Committee shall submit no more than 3 qualified nominees to The South Dakota Academy of Family Physicians Board of Directors, who shall act as a final Selection Committee.
   4. Award to be announced and presented at a time and occasion deemed appropriate by The SDAFP. Press releases will be conveyed to the news media.
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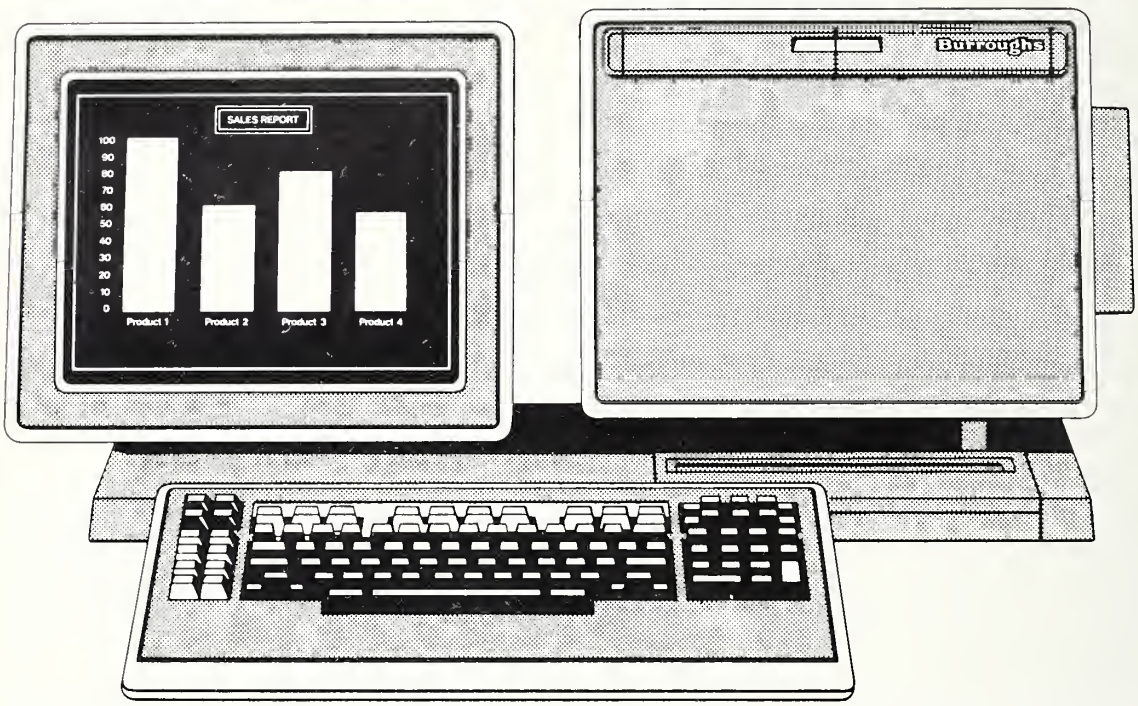

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1. To serve as an advisory committee to the auxiliary
2. To evaluate auxiliary programs and procedures, taking into consideration the present and future growth of the auxiliary
3. To focus on future objectives and make recommendations to stimulate decisions which would contribute to the growth of the auxiliary
4. To be aware of the resources of the auxiliary when making recommendations

The 1982-1983 American Medical Association Auxiliary Long Range Planning Committee recommends that the following goals be pursued for attainment by 1987-1988:

. . . to attain an AMA Auxiliary membership of 100,000;

. . . to achieve 100 per cent state representation within the national organization by means of a working structure within each state;

. . . to develop good working relationships between state and county auxilians and their medical societies and executives, including voting auxiliary representatives on selected medical society committees;

. . . to create a positive national image through innovative health-related projects;

. . . to make Auxiliary membership a unified membership;

. . . to stress the importance of legislative activity in health-related areas, and create an awareness of potential member impact upon local legislation;

. . . to continue to identify and service the needs of specific membership segments.

As we travel, each member holds the key to happy journeying for the community and the auxiliary. How you travel can make all the difference.

TRAVEL EXPECTANTLY. Expect adventure and success. Keep the auxiliary alive and growing by helping make decisions, weighing pros and cons, and supporting decisions of the majority.

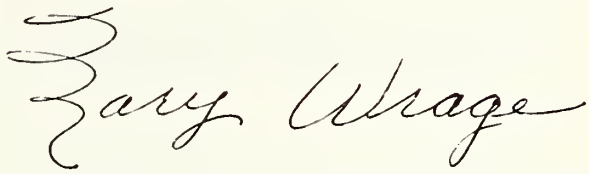
TRAVEL WITH CURIOSITY. It's not how far, but how deep you go that mines the gold of experience. Encourage expression of viewpoints, understand and exercise your membership rights, share in the welfare of your organization and shoulder your part of the load.

TRAVEL WITH IMAGINATION. Spark interest and involvement through your enthusiasm. Be positive. See the total picture. Be receptive to new ideas and goals.

TRAVEL PATIENTLY. Take time to understand others. Be flexible and open to people and situations. Help reconcile disagreements, mediate differences. Accept people's strengths, weaknesses.

TRAVEL FEARLESSLY. Banish worry, timidity, doubt. Know your strengths and weaknesses, as well as your potential. Have courage in convictions.

Equal commitment is vital to the success of our Auxiliary . . . won't you help us attain this commitment? Our fruitful family tree yields a bountiful crop.



Mary Wrage, President
South Dakota State Medical Association Auxiliary

P.S. November 6, 1984, Americans go to the polls to make their votes count. You know that more and more the way you practice medicine is determined by these government officials. In the last few years alone, critical issues such as Mandatory Assignment, Baby Doe, DRGs, Licensing, Hospital Staff Privileges and Third Party Reimbursement for non medical providers have been the focus of government activity.

Candidates look at where the votes come from . . . they know what one million votes means. But, if you and other members of our medical family are not registered and don't vote, the direction of our country and the medical professional will be shaped without your voice being heard. Making your vote count is part of responsible medical citizenship.

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Subscription $10.00 per year $1.50 per copy
Foreign $15.00

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ORIGINAL MANUSCRIPTS: Material appearing in all publications of the Journal of Medicine should be typewritten, double-spaced and the original copy, not the carbon should be submitted. An abstract of 100-200 words should accompany each scientific article. Footnotes should conform with the requirements for manuscripts, and each manuscript should include the name of the author, title of article and the location of the author. The used manuscript is not returned but every effort will be made to return manuscripts not accepted or published by the Journal of Medicine. Articles are accepted for publication on condition they are contributed solely to this Journal.

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Future Meetings

November

Pediatric Neurology and Neurosurgery, Mayo Mem. Aud. U. of Minn., Minneapolis, MN, Nov. 7. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Current Concepts in Gastrointestinal Pathology, Mayo Mem. Aud., Univ. of Minn., Minneapolis, MN, Nov. 9. Fee: $120. 6 hrs. Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Dementia: Dealing with Patient and Family Depression, Battenfeld Aud., U. of Kan. Med. Ctr., Kansas City, KS, Nov. 14. Fee: $30. 8 hrs. Category I credits. Contact: Jan Johnston, Off. of CME, U. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

December

Advanced Trauma Life Support, Orr Major Hall, U. of Kans. Med. Ctr., Kansas City, KS, Oct. 18-19. Fee: $350. 16 hrs. AMA Category I credits & 15 hrs. AAFP credits. Contact: Jody Scott, Dept. of Surg., U. of Kans. Med. Ctr., 39th & Rainbow, Kansas City, KS 66103. Phone: (913) 588-6124.

January

MRI, CT and Interventional Radiology, Fairmont Hotel, San Francisco, CA, Jan. 7-11. Fee: $425. 27 hrs. Category 1 credits. Contact: U. of Calif., San Francisco, Dept. of Radiology, San Francisco, CA 94143. Phone: (415) 666-5731.

Contemporary Issues in Obstetrics and Gynecology, Sheraton Hotel, Ixtapa, Mexico, Jan. 20-26. Fee: $200. 20 hrs. Category I credits; 18 hrs. AAFP credits. Contact: Jan Johnston, Off. of CME, Univ. of Kans. Med. Ctr., 39th & Rainbow Blvd., Kansas City, KS 66103. Phone: (913) 588-4480.

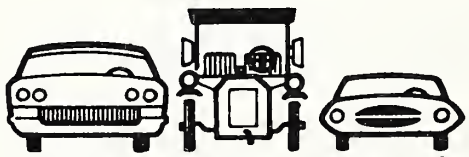
Recent Advances in Geriatric Medicine: Dementia in the Elderly, Holiday Inn, Embarcadero, San Diego, CA, Jan. 31-Feb. 2. Fee: $210. 13 hrs. CME credits. Contact: Off. of CME, UC San Diego School of Med., M-017, La Jolla, CA 92093. Phone: (619) 452-3940.

February

Diagnostic Radiology Seminars, The Gant, Aspen, CO, Feb. 2-9. Fee: $425. 21 hrs. Category I credits. Contact: U. of Calif., San Francisco, Dept. of Radiology, San Francisco, CA 94143. Phone: (415) 666-5731.

19th Annual Symposium on Cosmetic Surgery, Miami, FL, Feb. 7-9. Contact: Plastic Surgery Ed. Foundation, Exec. Offices, 233 N. Michigan Ave., Suite 1900, Chicago, IL 60601.

Immunology and Cancer, Shamrock Hilton, Houston, TX, Feb. 26-Mar. 1. Contact: Off. of Conf. Services, Box 131, M. D. Anderson Hosp. and Tumor Instit., 6723 Bertner Ave., Houston, TX 77030. Phone: (713) 792-2222.



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Before prescribing, see complete prescribing information in SK&F CO. literature or *PDR*. The following is a brief summary.

WARNING
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsivenss to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

Supplied: 'Dyazide' is supplied in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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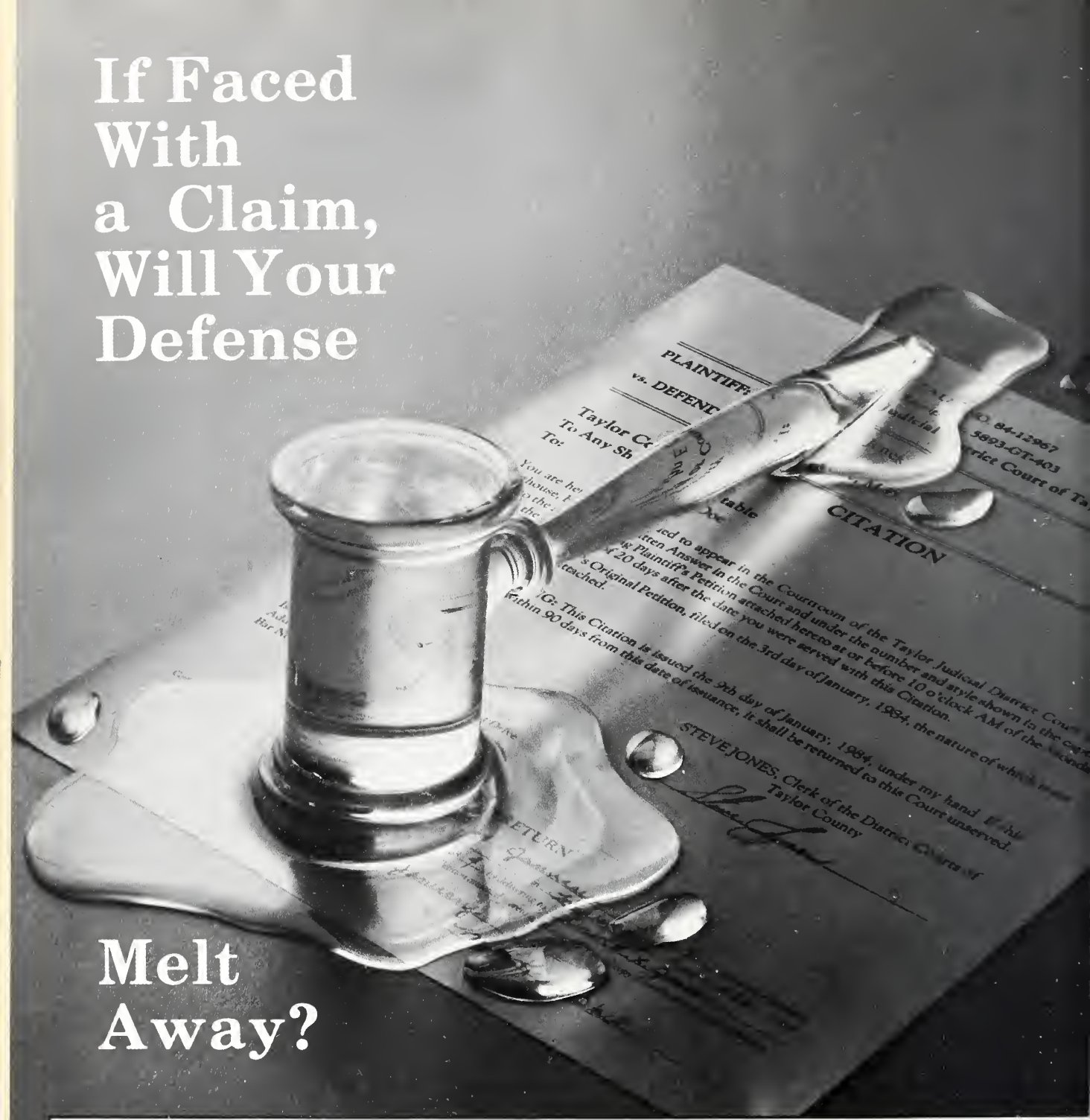

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ibuprofen

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Contraindications: Anaphylactoid reactions have occurred in individuals hypersensitive to *Motrin* Tablets or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin, iodides, or other nonsteroidal anti-inflammatory agents.

Warnings: Peptic ulceration and GI bleeding, sometimes severe, have been reported. Ulceration, perforation and bleeding may end fatally. An association has not been established. Use *Motrin* Tablets under close supervision in patients with a history of upper gastrointestinal tract disease, after consulting ADVERSE REACTIONS. In patients with active peptic ulcer and active rheumatoid arthritis, try nonulcerogenic drugs, such as gold. If *Motrin* Tablets are used, observe the patient closely for signs of ulcer perforation or GI bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity with papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with *Motrin* Tablets.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue *Motrin* Tablets and the patient should have an ophthalmologic examination, including central visual fields and color vision testing.

Fluid retention and edema have been associated with *Motrin* Tablets; use with caution in patients with a history of cardiac decompensation or hypertension. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of *Motrin* Tablets safety in patients with chronic renal failure have not been done.

Motrin Tablets can inhibit **platelet aggregation** and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and on anticoagulant therapy.

Patients should report signs or symptoms of **gastrointestinal ulceration** or bleeding, skin rash, weight gain, or edema.

Patients on prolonged **corticosteroid therapy** should have therapy tapered slowly when *Motrin* Tablets are added.

The antipyretic, anti-inflammatory activity of *Motrin* Tablets may mask inflammation and fever.

As with other nonsteroidal anti-inflammatory drugs, borderline elevations of liver tests may occur in up to 15% of patients. These abnormalities may progress, may remain essentially unchanged, or may be transient with continued therapy. Meaningful elevations of SGPT or SGOT (AST) occurred in controlled clinical trials in less than 1% of patients. Severe hepatic reactions, including jaundice and cases of fatal hepatitis, have been reported with ibuprofen as with other nonsteroidal anti-inflammatory drugs. If liver disease develops or if systemic manifestations occur (e.g. eosinophilia, rash, etc.), *Motrin* should be discontinued.

Drug interactions. *Aspirin:* used concomitantly may decrease *Motrin* blood levels.

Coumarin: bleeding has been reported in patients taking *Motrin* and coumarin.

Pregnancy and nursing mothers: *Motrin* should not be taken during pregnancy or by nursing mothers.

Adverse Reactions: The most frequent type of adverse reaction occurring with *Motrin* is gastrointestinal of which one or more occurred in 4% to 16% of the patients.

Incidence Greater than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence); **Central Nervous System**: Dizziness,* headache, nervousness; **Dermatologic**: Rash* (including maculopapular type), pruritus; **Special Senses**: Tinnitus; **Metabolic/Endocrine:** Decreased appetite; **Cardiovascular**: Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence less than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests; **Central Nervous System**: Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma; **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia; **Special Senses**: Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS); **Hematologic**: Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit; **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations; **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting; anaphylaxis; bronchospasm (see CONTRAINDICATIONS); **Renal**: Acute renal failure in patients with pre-existing significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria; **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence less than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis; **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri; **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions; **Special Senses**: Conjunctivitis, diplopia, optic neuritis; **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction; **Cardiovascular:** Arrhythmias (sinus tachycardia, sinus bradycardia); **Allergic**: Serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis; **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with *Motrin*. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under *"Probable Causal Relationship (PCR)"* if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under *"Causal Relationship Unknown"* if seven or more events have been reported but the criteria for PCR have not been met.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis. Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Do not exceed 2400 mg per day. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary.

Caution: Federal law prohibits dispensing without prescription.

MED B-7-S

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Upjohn The Upjohn Company
Kalamazoo, Michigan 49001

J-4791 August, 1984

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SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 10 October 1984 (ISSN 0038-3317)

Official Publication of the
South Dakota State
Medical Association

the South Dakota Chapter
of The American Academy
of Family Physicians

the South Dakota Chapter
of the American College
of Surgeons

the South Dakota
Psychiatric Association
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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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Ventilation and Perfusion Lung Scans, Sioux Valley Hospital-1981

Adenocarcinoma of the Endometrium

Clinicopathological Conference
Twenty-Eight Year Old Caucasian Female With Left Flank Pain and Renal Mass

December is

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Forty Year Old Man in Coma With Respiratory Distress After Falling Into a Hog Pit

Peter J. Erickson, M.D.*
David R. Rossing, M.D.†
Discussers

John F. Barlow, M.D.‡
Editor

CASE #1 013 936

This 40 year old Caucasian farmer was admitted to Sioux Valley Hospital in coma.

This patient had left to inspect a hog pit on his farm. When he did not return after approximately 20 to 30 minutes, the patient's son searched and found the patient in the hog pit partially submerged. The son did extract him from the hog pit but did not survive. Ambulance personnel noted tetanic movements, a pale color in the extremities with some central cyanosis, and bite marks around the tongue. He was in respiratory distress and was coughing up abundant frothy fluid and manure. He was given intravenous fluids, bicarbonate, cephalothin, and dexamethasone for his pulmonary and metabolic condition and diazepam for agitation. There was difficulty intubating the patient so that he was given oxygen by mask on his trip to the hospital. On admission he was noted to be in marked respiratory distress with central cyanosis and to have severe hypotension in the range of 60/0. He had many rhonchi in the chest. He was admitted to the Sioux Valley Hospital intensive care unit.

Intravenous therapy with dexamethasone was started. Initial blood gases were pH 7.19, pCO2 46 torr and pO2 38 torr. A Swan-Ganz catheter, intravenous line, arterial line, and endotracheal tube were placed.

PHYSICAL EXAMINATION: The patient was comatose initially and was gasping and coughing up bloody fluid. After stabilization his pulse was 122/minute and regular, respirations 20/minute with gasping and blood pressure (after intravenous therapy) 105 systolic and 64 diastolic. The patient responded to stimuli and was intermittently quite agitated but moved all four extremities. The pupils were constricted but responded to light. No other abnormalities of the head and neck were noted. Examination of the lungs showed scattered rhonchi and crackles at the right base. There was no wheezing. The heart had a regular rhythm with a grade II (six grades) holosystolic murmur best heard at the apex. The abdomen was soft and tender without palpable tenderness, organs or masses. The extremities were unremarkable and all pulses including the carotid pulses were 2-3+ and equal. The patient did respond to painful stimuli, moved all four extremities and had flexor-plantar responses.

HOSPITAL COURSE: After admission the patient underwent fiberoptic bronchoscopy which showed diffuse inflammation and hemorrhages but no foreign material. The day after admission the patient became increasingly alert with stable vital signs and pO2 74 torr, pH 7.32, and pCO2 42 torr on oxygen. He was treated on a ventilator with PEEP (positive end expiratory pressure). Initial chest film showed hazy infiltrates in the lungs consistent with pulmonary edema which worsened over the first 24 hours. On the second hospital day a 30% right pneumothorax was found. A right chest tube was inserted. There was subsequent disappearance of the pneumothorax but subcutaneous emphysema appeared and the pulmonary infiltrates persisted. Bilateral areas of consolidation tended to concentrate in the lower lobes. The infiltrates cleared slightly toward the latter part of his course.

On the 12th and 13th hospital day the patient developed diarrhea and became lethargic. He had been febrile to about 101°F but his temperature rose to between 102°F and 103°F. He also developed a diffuse rash which was described as erythematous and macular with blanching. This was felt to be secondary to a drug reaction. The patient became progressively lethargic with intermittent agitation and continued to be febrile. There was some hyperflexia with an

* Resident in Family and Community Medicine, Program Affiliated with School of Medicine, USD, Sioux Falls, SD.
† Pulmonologist, Central Plains Clinic, Assistant Professor of Medicine, School of Medicine, USD, Sioux Falls, SD.
‡ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, School of Medicine, USD, Sioux Falls, SD.

extensor plantar response on the left. An initial computer tomogram of the brain (CT) was negative and left maxillary sinus films showed mucosal thickening. However, subsequent CT scan of the brain revealed bilateral hemispheric lesions consistent with abscesses. On the 27th hospital day a right frontal burr hole and tapping of a large brain abscess was performed.

LABORATORY DATA: Initial urinalysis was yellow, clear, specific gravity 1.013, pH 5.0 with 2+ protein, negative glucose, ketones and bilirubin and 3+ hemoglobin; sediment 2-3 white cells/hpf, 60-75 red cells/hpf and 0-3 finely granular casts/lpf. Subsequently urinalyses show a disappearance of the protein but persistence of the hemoglobin and red cells in the urine. There was no significant pyuria. The patient had a catheter in place. Initial hemoglobin was 18.9 gm/dl, but this declined over the hospital course progressively (partly due to hydration) to a low of 11.7 gm/dl. Other red cell parameters were not significantly altered. The white count initially was 1.7×10^9/L but this rose progressively associated with the patient's fever to 14.9×10^9/L. There was a persistent neutrophilic leukocytosis with shift to the left early in the course. Platelet counts varied from an admission level of 167×10^9/L dropping to 93×10^9/L but finally rising along with the white count to levels exceeding 600×10^9/L. The blood urea nitrogen and creatinine were within normal limits. Multiple determinations of plasma electrolytes showed intermittent mild hyponatremia and initial low carbon dioxide content values. Initial blood gases showed metabolic acidosis with a pH as low as 7.19 and pO2 as low as 38 torr. The pH, CO2 content, and pO2, however, constantly improved. During the patient's febrile period, he developed respiratory alkalosis with a pH as high as 7.52 and pCO2 as low as 27 torr. The pO2 values and O2 saturation values were slightly below normal on rare occasions. The majority of blood gas determinations, however, were within normal limits.

Over the hospital course the patient had elevated lactic dehydrogenase (LD) values at 472 and 480 IU/L (0-70 IU/L). Total protein and calcium levels were slightly depressed and glucose levels were slightly elevated (the latter probably due to intravenous therapy with glucose containing solutions). A screen for disseminated intravascular coagulation was negative. Multiple blood and urine cultures revealed no growth. Escherichia coli and Proteus mirabilis were grown from the sputum. Stool culture and screen for ova and parasites in the stool were negative. Later in the course of the disease, Pseudomonas aeruginosa and Escherichia coli were grown from the sputum. Electrocardiogram showed sinus tachycardia during the patient's febrile episodes but otherwise were unremarkable. The patient was studied for arterial insufficiency of the upper and lower extremities but no definite abnormality was found. The patient was initially treated with ampicillin and tobramycin but was switched to clindamycin and cefotaxime. The patient also later received tobramycin and nafcillin. Moxalactam and chloramphenicol were added when the patient was found to have a brain abscess. The patient was transferred to the Mayo Clinic.

DR. ERICKSON: The most striking and important historical aspect of this patient's initial picture was that he was found submerged in a hog pit. To a city person like myself, the significance of this fact may not be immediately obvious but it is the most important clue as to the nature of the patient's problem.

This patient is suffering from an increasingly more common entity known as dung lung. In general this entity has four main aspects. 1) The direct effect of inhalation of toxic gases, 2) asphyxia, 3) aspiration of manure or other foreign material, and 4) infection from aspiration and being bathed in material rich in microbial flora.

To understand the clinical problem, one has to understand the nature of a hog pit. For various reasons, such as sewage disposal regulations and a desire for cheap recycled fertilizer, many farmers are now using liquid manure storage tanks or pits to collect and store the liquid manure that is washed out of the livestock barns. This liquid manure is allowed to sit and decay for some time before it is pumped out and spread over the fields as fertilizer. As this fecal soap is decaying, a variety of toxic gases are being produced from this rich organic mixture. More and more patients are developing considerable pulmonary problems when they accidentally or voluntarily enter these storage depots of liquid manure and inhale the vapors.

Let me review some of the gases that are formed in these pits. The major toxic gas is hydrogen sulfide (H_2S). This gas gives off a characteristic malodorous smell described as the odor of rotten eggs. Our noses are so sensitive to this that we can detect H_2S in a concentration as little as 0.2 ppm (parts per million). H_2S is an active cellular poison which like cyanide inhibits the cytochrome oxidase system of the cell leading to paralysis of aerobic metabolism and severe metabolic acidosis due to lactic acid production, a consequence of cellular hypoxia, and eventually to cell death.

A significant danger of H_2S is that at concentrations of 150 ppm there is complete paralysis of the sense of smell giving victims a false sense of safety as they enter the danger area.

This gas is so toxic that sometimes even if a victim is removed within a few minutes, the patient will still not be able to survive. At concentrations of 250 ppm the eyes and nose will water and there is irritation to the mucous membranes with the development of bronchitis and pulmonary edema. At 500 ppm there is central nervous system toxicity with headache, nausea, weakness, disorientation and loss of consciousness. Higher levels still will cause death very rapidly. One can easily detect this gas by the use of a simple lead acetate paper test.

Ammonia (NH_3) is another toxic gas generated from liquid manure. This gas is very irritating to mucous membranes and can produce pulmonary edema. Other gases produced include methane and carbon dioxide which displace oxygen in the air and produce an atmosphere low in oxygen concentration. In this way they tend to produce asphyxia by lowering the concentration of inspired oxygen.

Another deadly gas which can be produced from this swampish brew is carbon monoxide. This well known poison displaces oxygen from hemoglobin with the formation of carboxyhemoglobin and shifts the oxygen dissociation curve to the left. Both of

these effects of carbon monoxide produce severe tissue hypoxia.

With this knowledge we can perhaps reconstruct what happened to our patient. As he approaches the pit, he may have had some warning by detecting the odor of rotten eggs due to H_2S but as he approaches nearer into the higher concentration of H_2S, the odor goes away. He becomes disoriented, stumbles, gasps for air; and, perhaps, as he is becoming unconscious, he falls into the enteric soup inhaling it into his lungs further compromising ventilation which is already impaired from pulmonary edema and irritated swollen bronchial mucosa. The aspiration of the liquid manure further diminishes air exchange and exposes his lungs to a variety of infectious agents. At the same time his whole body is being covered by a rich microbial ointment. If the patient is rescued in time, he is hypoxic with lactic acidosis, pulmonary edema, and will probably go on to sepsis with pneumonia or other infections. He may suffer the effects of hypoxia on the central nervous system or other organ systems with seizures and coma.

Diagnosis of H_2S poisoning is made by history of recent collapse and can be confirmed by determination of sulfide levels in the blood of victims or in the air in the area of the hog pit. Levels of carboxyhemoglobin will also be helpful in managing the patient. There is usually severe metabolic acidosis due to a lactic acid formation typical of acute cellular hypoxia. Hypotension and hemoconcentration, which are common, are often due to dehydration and will respond to intravenous fluids. The adult respiratory distress syndrome (ARDS) is a direct effect of the inhalation of the toxic gases and the aspiration of the liquid dung. The patient is best treated, as he was in this case, with cardiopulmonary resuscitation, followed by mechanical ventilation with PEEP and oxygen. The presence of Escherichia coli, Pseudomonas aeruginosa and Proteus mirabilis organisms in the sputum as well as the further development of brain abscesses are all consistent with infectious agents one might expect in decaying manure.

An additional treatment which has been advocated is nitrites which inactivate sulfide and protect the cytochrome oxidase system allowing aerobic respiration. Methemoglobin production occurs with this agent; and, therefore, an excess of this material can impair oxygen transport.

In summary, entering a liquid manure storage area is not safe even if you cannot smell the gas, especially if air circulation in the area is poor or the tank has recently been agitated. The use of a safety line and self contained breathing apparatus as well as the presence of another observer when entering these areas is important. Rescue should be quick but the rescuers should also heed the above precautions as they also may become victims of the noxious gases. Multiple victims have been reported several times in these accidents at hog pits. I am not sure what caused the brain abscesses in this case but they could be due to a variety of organisms which I am sure Dr. Barlow will discuss.

Dr. Peter Erickson's Diagnoses
Dung Lung

DR. BARLOW: The patient was transferred to Mayo Clinic after aspiration of the brain abscess where he was treated with intravenous miconazole, chloramphenicol and cefazolin for the organism found. He continued to be febrile, comatose and required a tracheostomy. The brain abscesses by CT became enlarged. He was transferred back to Sioux Valley Hospital and then to his local hospital in Armour, S.D., where he died (Fig. 1).

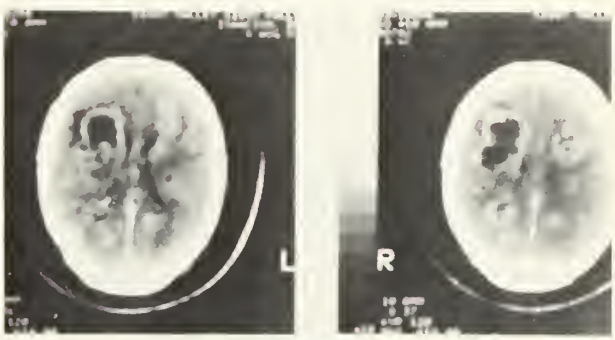

Figure 1
Computer tomogram showing large cavities due to brain abscesses.

The material from the brain abscess was cultured but no material for histologic examination was submitted. Within two days of incubation at 37°C on sheep blood, eosin-methylene blue (EMB), enriched chocolate agar and at 25°C incubation on Sabouraud's agar, there was abundant growth of a raised mycelial fluffy colony with a white periphery and central gray areas. Scotch tape preparation showed septate hyphae with terminal pyriform conidia (lollypops). Because we were uncertain of the diagnosis, the organism was referred to the Mycology Section of the Mayo Clinic where a diagnosis of Pseudallescheri boydii was made (Fig. 2).

This fungus has a complex nomenclature. The imperfect or asexual form, Monosporium apiospermum, first isolated, was given the name Allescheria boydii, when ascocarps indicating sexual reproduction were discovered, in order to designate the perfect form of the fungus. Petriellidium boydii is a newer name and this has been followed by Pseudallescheria boydii. The organism is a true fungus found in the soil of many parts of the United States. It has been the most common agent in the eumycotic

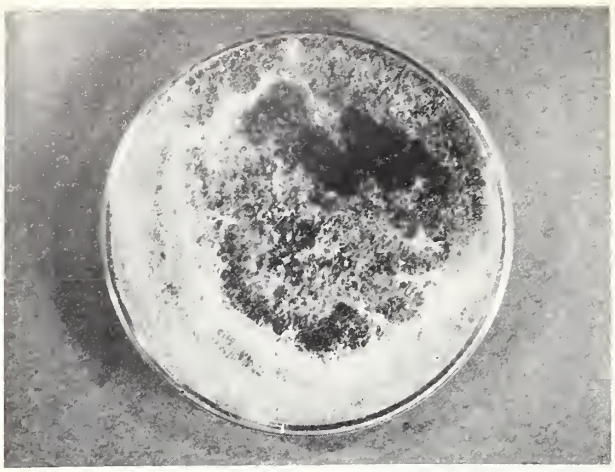

Figure 2
Typical culture of Pseudallescheria boydii obtained from brain abscess.

form of maduramycosis, a mycetoma characterized by multiple deep abscesses of soft tissue and bone with draining sinuses. Maduramycosis is more common in males probably due to the fact that it is acquired by trauma. In recent years this fungus has been a rare cause of visceral infections usually in immunocompromised hosts although a few patients without known immune defects, such as this patient, have acquired infection by this fungus. Beside posttraumatic bone and soft tissue infections, endocarditis, sinusitis, meningoencephalitis, as well as brain, thyroid, renal and prostate abscesses have been described. Both necrotizing pneumonia as well as saprobic growth in a previous pulmonary cavity (''fungus ball'') have been described. Parotitis, otomycosis and endophthalmitis have also been reported. The fungus tends to invade blood vessels similar to aspergillus and the zygomycetes (mucormycosis).

It is quite important to culture the organism since it cannot be distinguished histologically from Aspergillus. As in this case, it does grow rapidly on routine media used for bacterial and fungal isolation. However, although the organism is easy to isolate from tissue, sputum has not been a good specimen for isolation of Pseudallescheria boydii. Serologic techniques are also available for diagnosis in reference laboratories.

A very important reason for prompt diagnosis is that the organism does not respond to amphotericin B or 5 fluorocytosine. Miconazole and, perhaps, ketoconazole show promise in treatment of infections produced by this fungus. Surgery has been promoted especially in localized pulmonary infections and amputation may be necessary to eliminate maduramycosis of an extremity.

FINAL ANATOMIC DIAGNOSES

1. ACUTE ALVEOLAR DAMAGE SYNDROME (DUNG LUNG)

2. BRAIN ABSCESSES DUE TO PSEUDALLESCHERIA BOYDII

Diffuse alveolar damage (DAD) is a descriptive term for a nonspecific sequence of events following acute lung injury caused by any one of a variety of toxic agents producing damage of alveolar epithelial and endothelial cells.

Causes of DAD:

1. Infections — virus, mycoplasma
2. Inhalants — oxygen toxicity, nitrogen dioxide, sulfur dioxide, smoke and other fumes.
3. Drugs and ingestants — azathioprine, BCNU, bleomycin, busulfan, chlorambucil, cytosine arabinoside, mitomycin, cyclophosphamide, melphalan, methotrexate, colchicine, gold, hexamethonium, hydrochlorthiazide, nitrofurantoin, placidyl, practolol, heroin, kerosene, paraquat.
4. Shock — traumatic, hemorrhagic, septic, neurogenic, cardiogenic
5. Radiation
6. Miscellaneous — pancreatitis, cardiopulmonary bypass, air embolism, near drowning, uremia, high altitude, postlymphangiography, heat, molar pregnancy, systemic lupus erythematosus.

DAD is the underlying pathology of adult respiratory distress syndrome (ARDS) — Other synonyms used for ARDS are shock lung, posttraumatic pulmonary insufficiency, traumatic wet lung, congestive atelectasis, progressive pulmonary consolidation, adult hyaline membrane disease, and respirator lung.

Three stages of DAD occur:

1. An acute exudative phase characterized by edema, protein exudation and hyaline membranes in the alveoli with fibrin thrombi in small arteries is seen in the first week after the injury.
2. A proliferative or organizing phase with interstitial inflammation and fibrosis with often intense lining cell hyperplasia due to proliferation of type II pneumocytes follows.
3. Progression to diffuse interstitial fibrosis or honeycomb lung may follow. Resolution may occur depending on the extent and duration of the injury.

There is considerable overlap between the three stages and the course of the disease may not be complete for several weeks when a picture indistinguishable from usual interstitial fibrosis (UIP) is not an infrequent outcome.

A common sequela of DAD is bronchopulmonary

dysplasia in premature infants which, in part, may be due to oxygen toxicity.

The pathogenesis of DAD has been suggested to be direct lung injury as from blast or chest wall contusion, vasoactive peptides released from damaged tissue, microemboli or microthrombi of platelets and leukocytes either from infused unfiltrated blood or secondary to disseminated intravascular coagulation which produces fibrin thrombi. These thrombi cause capillary blockade or release platelet factors or leukocyte enzymes eventuating in tissue damage. Fat emboli, sepsis, acidosis and overhydration associated with acute respiratory insufficiency produce organ failure of the central nervous system, gastrointestinal tract, hepatic and renal systems. Multiple organ failure is common in bacteremic and nonbacteremic sepsis with or without disseminated intravascular coagulation.

Dr. Rossing, would you like to make a few comments?

DR. ROSSING: The diagnosis of the patient's acute pulmonary condition was, as suggested, adult respiratory distress syndrome or ARDS. He was treated appropriately with PEEP but developed a pneumothorax which is a common complication of this therapy. It was fortunate that he was able to be weaned from the respirator within a week. This was probably due, in part, to the fact that he was an otherwise healthy man with excellent organ system function. The antibiotics mentioned were given at various times and combinations during his prolonged hospital stay. A significant problem in treating patients with ARDS is that it is difficult to know when they develop a pneumonia. This is especially true since their upper respiratory tract becomes colonized with gram negative rods and their chest x-ray continues to show infiltrates. One attempts to base the therapy on cultures and the patient's clinical status. The changes in antibiotic coverage in this case reflect this approach.

The development of his brain abscesses first manifested themselves as an insidious progressive lethargy. As was suggested, he had a remarkably blunted affect considering the tragedy which he had recently experienced, and, the possibility of severe depression was entertained. For this reason, a psychiatric consultation was sought. Only when the patient became febrile was the diagnosis of central nervous system infection suspected. As was stated, the first CT of the brain was negative but subsequent CT's showed the developing abscesses.

* DR. JOHN MALM: Nitrofurantoin was mentioned as a cause of ARDS. Is this common and can it occur on a short course of therapy?

DR. ROSSING: While ARDS has been reported, in my experience it is rather uncommon with this drug. An acute allergic hypersensitivity pulmonary reaction is not uncommon and a fibrosing alveolitis may develop with long-term ingestion. This is one reason why we tend to discourage the use of nitrofurantoin in patients with urinary tract infections susceptible to other antibiotics such as sulfonamides or ampicillin.

I would also like to mention that the term ARDS is controversial. There are now three centers in the United States funded for a five year National Institutes of Health (NIH) study of ARDS in Boston, San Antonio, and San Diego. The investigators in these centers are trying to determine the cellular basis of this acute lung injury.

Again I would like to stress the insidious problem of infection in ARDS. If the patient develops a leukocytosis or fever, a thorough study for a hidden source of sepsis is indicated.

* Family Practitioner, Gregory, SD.

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By the time this page is printed, the general election of 1984 will be history. I hope and assume that all of our SDSMA members have utilized their privilege of voting. No matter how the election comes out, we must continue our diligent pursuit of maintaining the highest quality of care for our patients.

It is obvious that the federal bureaucracy relates all health care to the basis of cost. Unfortunately, this can mean that eventually, and in the not too distant future, we will be faced with rationing of health care. It will be interesting to see who becomes the "rationer" and who will be the "rationee." An excellent article well worth reading is printed in the "My Opinion" section of the September 21, 1984 issue of AMERICAN MEDICAL NEWS by William S. Weil, M.D., president of the Los Angeles County Medical Association. We must continue our dialogue with our state and federal elected officials, who, after all, really are our employees. Our best means of being heard and actually listened to is through our AMA. Our federation has the organization and expertise to be the spokesman for medicine in America. If you are not now an AMA member, I urge you to become an active member to insure the future of the private practice of medicine in our great land.

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PRESCRIBED CREDIT VS. CATEGORY I CREDIT

Explanation: In the early years of the American Academy of Family Physicians, the designated terms for approved continuing medical education courses/programs were Category I and Category II. To avoid confusion with the "later" American Medical Association categories, the Academy changed its terminology to "Prescribed" and "Elective," with the wish to differentiate its programmatic approval from the AMA's institutional approval.

PRESCRIBED CREDIT (AAFP) is defined as the credit awarded for each program which has family practice (Academy member) input to assure relevance to family practice and for which sponsors/producers have obtained approval from the AAFP Commission on Continuing Medical Education prior to the program date.

CATEGORY 1 (AMA) has the definition: "An activity can be designated AMA Category I if it is provided or cosponsored by an institution or organization accredited for continuing medical education by ACCME or by a state medical society. . . ." (ACCME is the acronym for the Accreditation Council for Continuing Medical Education.)

Basically, the difference between AAFP Prescribed credit and the AMA Category I credit is that EACH AAFP course/program has family practice input to assure that the program is relevant to family practice and is individually reviewed by the COCME; the AMA Category I credit is awarded through an institution which offers acceptable continuing medical education.

For any program offered by an institution accredited by the ACCME, an Academy member is automatically eligible for AAFP Elective credit. Only those programs individually reviewed and approved for Prescribed credit by the AAFP COCME, and/or through its regional advisors, are entered as Prescribed credit on a member's record.

COMMITMENT TO IDEALS

Dr. Edmund D. Pellegrino, director of the Kennedy Institute, Georgetown University, Washington, D.C., speaking at a recent S.T.F.M. meeting, applied the need for commitment to ideals beyond family practice, to the entire medical profession — the "family of medicine."

He said that a family is united by a common set of ideals, and that he had observed a "central disquiet" in medicine that "our moral cement — dedication to ideals — is loosening."

It is generally accepted that all physicians are bound by their education, an interest in patients and commitment to good care, Dr. Pellegrino said.

However, he added, "We're diverse. We can no longer assume that a common interest in patients will bind us."

He cited differences in approach to medical procedures and to how health care dollars should be spent.

"We are a pluralistic profession in a pluralistic society," he said, "and pluralism without common values is pluralism gone berserk."

"You in family medicine confronting illness may have a special role in building the family of medicine," he challenged his audience.

He described three views of medicine: (1) the physician primarily a technician, with actions pegged to the medical good; (2) medicine primarily a means of livelihood; and (3) medicine primarily a helping and healing profession.

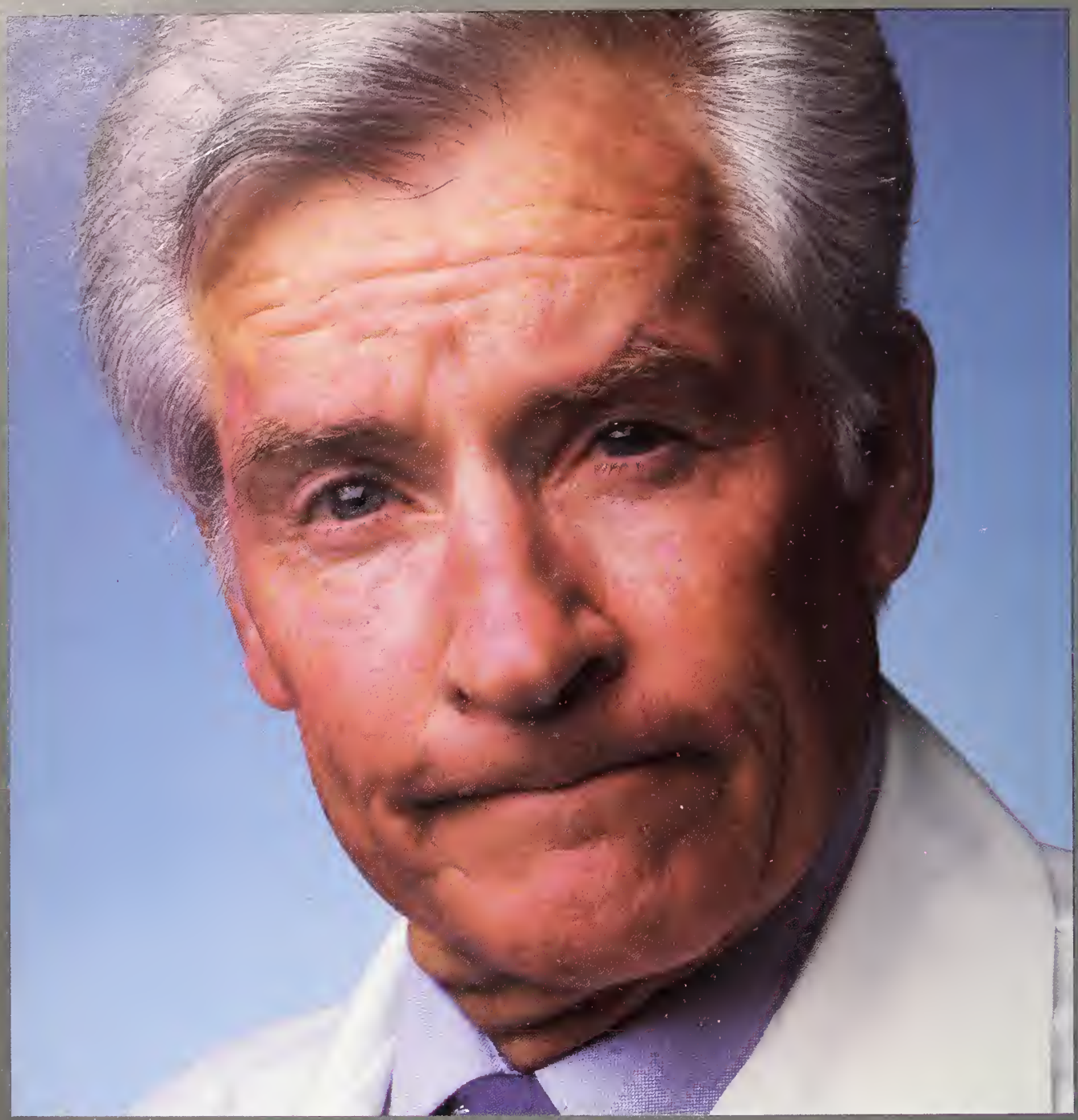
"I am hoping we can make a statement of commitment to the helping and healing concept," he said, adding that the public is repulsed by the first two views. "In due course, the public will find us out — they always do," he said.

"Medicine has been one of the noblest professions but cannot remain so unless we are devoted to noble commitments," he said.

Reiterating the special challenge, Dr. Pellegrino said, "Family medicine, if true to its devotion, can only choose the healing and helping model. Therefore, it has a role in reformation of medicine — both in the nuclear family [its specialty] and the extended family [all of medicine]."

USDSM M.D. Graduates AAFP
(Specialty Society) Student Membership

Academic Year	Number of Graduates	Number of Student Members	Number of Student Members by Third Year Clerkship Base		Number of Student Members Entering F.P. Residencies By Third Year Clerkship Base	
			X	Y	X	Y
1976-77	39	24	16/26	8/13	7/16	0/8
1977-78	36	23	15/23	8/13	4/15	0/8
1978-79	39	21	15/27	6/12	6/15	2/6
1979-80	47	25	17/31	8/16	6/17	2/8
1980-81	49	26	18/33	8/16	9/18	3/8
1981-82	44	22	18/29	4/15	1/18	0/4
1982-83	49	28	20/31	8/18	3/20	3/8
1983-84	50	25	17/34	8/16	4/17	1/8
	353	194	136/234 (58%)	58/119 (49%)	40/136 (29%)	11/58 (19%)
			194/353(55%)		51/194(27%)	



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Management of Retained Common Bile Duct Stones

John B. Marshall, M.D.*
Kenneth A. Vogele, M.D.†

ABSTRACT

Retained common bile duct stones are a fairly common clinical problem and are associated with significant clinical and operative morbidity and mortality. This paper reviews the problem, their management and prevention. The relatively new technique of endoscopic sphincterotomy is discussed. This procedure has probably supplanted surgical re-exploration and treatment of most retained common duct stones.

Common bile duct stones are present in about 15% of patients undergoing cholecystectomy for cholelithiasis.[1, 2] Reoperation will be required in 2-10% of patients for residual or recurrent common bile duct stones.[2, 3] Retained choledocholithiasis are associated with significant clinical and operative morbidity and mortality. This paper will review their management and prevention. Emphasis is given to the new technique of endoscopic retrograde sphincterotomy which is an effective nonoperative method of removing retained common bile duct stones.

INCIDENCE

There are about 625,000 cholecystectomies performed in the United States annually.[4] Of this number, it is estimated that 93,750 patients (15%) will undergo common bile duct exploration. Of this group, about 9,500 will have a negative exploration. At the time of postoperative T-tube cholangiography, about 14,000 patients (15% of 93,750) will be found to have residual stones. Since about 31,250 patients (5% of 625,000) with no suggestive clinical history will have unsuspected stones within the common duct (which would be undetected without routine operative cholangiography), there will be a potential population in excess of 20,000-40,000 patients who will leave the operating room yearly with stones remaining in the common duct.[4]

* Staff Gastroenterologist, Rapid City Regional Hospital, Rapid City, SD.
† Staff Gastroenterologist, Rapid City Regional Hospital, Rapid City, SD.

CLINICAL PRESENTATION

Choledocholithiasis may cause no symptoms. They may be present for months or years before causing problems. The presentation of common duct stones may take several forms, including: (1) Biliary colic, (2) Jaundice and liver test abnormalities, (3) Cholangitis, and (4) Pancreatitis.

AVOIDING RETAINED COMMON DUCT STONES: THE IMPORTANCE OF ROUTINE OPERATIVE CHOLANGIOGRAPHY

A major goal of surgeons performing cholecystectomy is to ensure removal of common bile duct stones in order to avoid later obstructive complications. The routine performance of operative cholangiography in association with cholecystectomy reduces the incidence of retained common bile duct stones. Unsuspected stones are found in 5% of instances.[4, 5]

Common bile duct exploration (and placement of a T-tube) is performed when choledocholithiasis are suspected. It is not performed routinely because of increased operative morbidity and mortality. Absolute clinical indications for exploring the bile duct include palpable stones in the duct and cholangitis. Relative clinical indications include jaundice, pancreatitis, a dilated duct, and small stones in the gallbladder. Use of operative cholangiography as an adjunct to these indications for common bile duct exploration has resulted in a decrease in negative

exploration from 40 to 10%.[6] In addition to removing any found stones, the surgeon usually performs a drainage procedure (such as sphincteroplasty) when the duct is markedly dilated, when more than about five stones are found, when all the stones cannot be removed, or when there have been one or more previous operations for common duct stones.[1]

Optimal operative cholangiography requires close collaboration between surgeon and radiologist, the best equipment available, as well as careful attention to technique. This has recently been reviewed.[4]

Some surgeons still question the cost effectiveness of operative cholangiography. What is often overlooked is the total cost for the care of the patient who has retained stones. To the cost of the initial surgery must be added the expense of later corrective measures.[4] High quality routine operative cholangiography during cholecystectomy helps reduce the incidence of retained biliary stones.

NONOPERATIVE EXTRACTION OF RETAINED STONES WITH T-TUBE IN PLACE

After common bile duct exploration, a T-tube is inserted and the duct closed. The routine use of at least a 14 French T-tube is recommended, even if no retained stones are suspected. The T-tube can be removed 10-12 days after surgery if a cholangiogram through the T-tube is negative. If retained stones are detected on the post-operative T-tube cholangiogram, the T-tube tract can later be used for non-operative stone extraction under roentgenographic control. This technique carries considerably less morbidity and mortality than surgical re-exploration and can be done, in many cases, as an out-patient.

Patients with retained common duct stones and a T-tube in place, report to the Radiology Department 4-5 weeks post-operatively. This interval of time allows the spontaneous passage of some stones. It also allows time for a mature sinus tract to form which can be used for stone extraction. After the location of any retained stones is ascertained on a repeat T-tube cholangiogram, the T-tube is withdrawn. A steerable basket catheter is inserted through the sinus tract into the bile duct and stone extraction performed. Occasionally, balloon catheters are used. Large stones (1 cm or larger) usually require fragmentation unless a very large T-tube has been inserted. Dilation of the sinus tract may be required to extract large stones. Occasionally, multiple sessions are required to remove all stones. Stone dissolution with monooctanoin infused through the T-tube can also be tried in difficult cases.[7]

In experienced hands, the success rate for non-operative extractions is about 95%.[8] In a series of 631 cases, there was no mortality and a morbidity of about 5%.[9] Prophylactic antibiotics are not used routinely.

In patients in whom T-tube tract stone extraction is unsuccessful, endoscopic removal (mortality of about 1%) is generally preferred over reexploration (mortality about 3%).

REMOVAL OF RETAINED COMMON DUCT STONES WHEN THERE IS NO T-TUBE

Until recently, re-exploration has been the only technique available to remove retained common duct stones without a T-tube in place. Over the past ten years, the technique of endoscopic sphincterotomy has been developed as a non-operative method of removing common bile duct stones at the time of retrograde cholangiography.[2, 10, 11]

Endoscopic sphincterotomy is undertaken by an accomplished endoscopist with extensive retrograde cholangiopancreatographic experience. The procedure is done in a fluoroscopy room using a side viewing duodenoscope with the patient sedated. The sphincterotome knife consists of a plastic catheter containing a wire, the distal 30 mm of which is externalized at the catheter tip. Selective cannulation of the common bile duct is performed with the sphincterotome passed freely into the common bile duct (Figure 1). The wire is then pulled taut in about a 12 o'clock position to form a cutting bow (Figure 2). A blended cutting current is used to effect a 10-15 mm cut through the ampullary sphincteric mechanism. Many stones will pass spontaneously after an adequate sphincterotomy cut has been made. The remainder can usually be extracted with an endoscopically inserted balloon catheter or dormia basket.

Endoscopic sphincterotomy has a success rate of 80-95%, with a complication rate of about 7% and mortality of 1-1.5%. This compares very favorably with re-exploration and has the additional advantage of requiring a much shorter hospital stay and being less costly. Large stones (greater than 2-2.5 cm.) may be a relative contraindication to doing the procedure.

Endoscopic sphincterotomy has probably supplanted surgical re-exploration as treatment of most retained common duct stones (in patients with a previous cholecystectomy). It can also be considered in high risk patients with common bile duct stones and an intact gallbladder.

Recently another non-surgical approach to the management of retained common bile duct stones has been developed,[12] that of percutaneous transhepatic stone extraction, but experience with this technique is limited.

FIGURE 1
Endoscopic cholangiogram demonstrating retained common bile duct stones (see case report). Sphincterotome is freely cannulated within the common bile duct.

FIGURE 2
Schematic sketch of endoscopic sphincterotomy. The sphincterotome is in the distal common bile duct. The wire is pulled taut in about a 12 o'clock position to form a bow which will cut the roof of the papilla. (Used with permission: CLINICS IN GASTROENTEROLOGY, Jan. 1983, p 510).

INITIAL EXPERIENCE WITH ENDOSCOPIC RETROGRADE SPHINCTEROTOMY AT THE RAPID CITY REGIONAL HOSPITAL

We started doing endoscopic retrograde sphincterotomy (ERS) at the Rapid City Regional Hospital in September of 1983. Between September of 1983 and July of 1984, seven patients presented with a clinical picture suggestive of choledocholithiasis and were felt to be candidates for ERS. The seven patients ranged in age from 40 years to 90 years with a mean age of 72.6 years. Five were 75 years of age or older. Six of the seven patients had had a previous cholecystectomy. One patient had an intact gallbladder, but was considered for ERS because of high operative risk in view of severe rheumatoid arthritis and malnutrition. ERS was able to be performed in 6 of the 7 patients (85.7%) and was successful in all six. In the seventh patient, the sphincterotome was not able to be freely passed into the common bile duct and it was elected not to attempt the procedure. ERS was performed at the time of the initial endoscopic retrograde cholangiography (ERC) in five of the patients. The length of hospitalization attributable to ERC, ERS, and bile duct stone extraction averaged three to four days. ERS resulted in no complications in our patients.

ILLUSTRATIVE CASE REPORT: ENDOSCOPIC SPHINCTEROTOMY

A 78 year old man was admitted to the hospital with upper abdominal pain and jaundice.

He had been hospitalized 18 months previously with an episode of right upper quadrant abdominal pain and vomiting. Mild liver test abnormalities were present at that time including bilirubin 1.9 mg/dl (normal, 0.2-1.2), SGOT 62 IU/L (normal, 6-41), and alkaline phosphatase 246 IU/L (normal, 38-120). Evaluation included a nonvisualizing oral cholecystogram. Other studies included negative upper abdominal ultrasound exam, upper GI x-ray series, and barium enema. A laparotomy with cholecystectomy was done with the finding of cholelithiasis. Operative cholangiography was not done.

The patient did well after his cholecystectomy until 5 days prior to admission when he developed steady right upper quadrant abdominal pain associated with nausea and vomiting. Three days later the patient was noted to be jaundiced. Admission laboratory tests included a WBC count 8100/cu mm, with a slight left shift, and hemoglobin 14.2 g/dl. Liver tests were abnormal including bilirubin 9.3 mg/dl, SGOT 153 IU/L, and alkaline phosphatase 656 IU/L. Serum amylase and lipase were both elevated to about twice the upper limit of normal. The

patient's clinical and lab picture supported a diagnosis of common bile duct stones with biliary stone-induced pancreatitis. Endoscopic retrograde cholangiography was performed with the finding of two common bile duct stones (Figure 1). Anticipating this finding, endoscopic retrograde sphincterotomy was performed immediately thereafter and the stones removed. Post hospitalization he did well and his liver tests normalized. Outpatient endoscopic retrograde cholangiography was performed 6 weeks later and showed a widely patent sphincterotomy opening. The follow-up cholangiogram showed no residual biliary stones (Figure 3).

FIGURE 3
Endoscopic cholangiogram (same patient as Figure 1) done after sphincterotomy procedure and demonstrating disappearance of stones.

COMMENT: The performance of routine operative cholangiography at the time of his cholecystectomy might well have avoided his later problems with common bile duct stones. Endoscopic retrograde sphincterotomy provided a relatively simple, safe, and cost efficient way of removing this elderly gentleman's stones.

CONCLUSIONS

Retained common duct stones continue to be a fairly common clinical problem associated with significant clinical and operative morbidity and mortality. The routine performance of high quality operative cholangiography during cholecystectomy helps reduce the incidence of retained biliary stones. The technique of endoscopic sphincterotomy has probably supplanted surgical re-exploration and treatment of most retained common duct stones.

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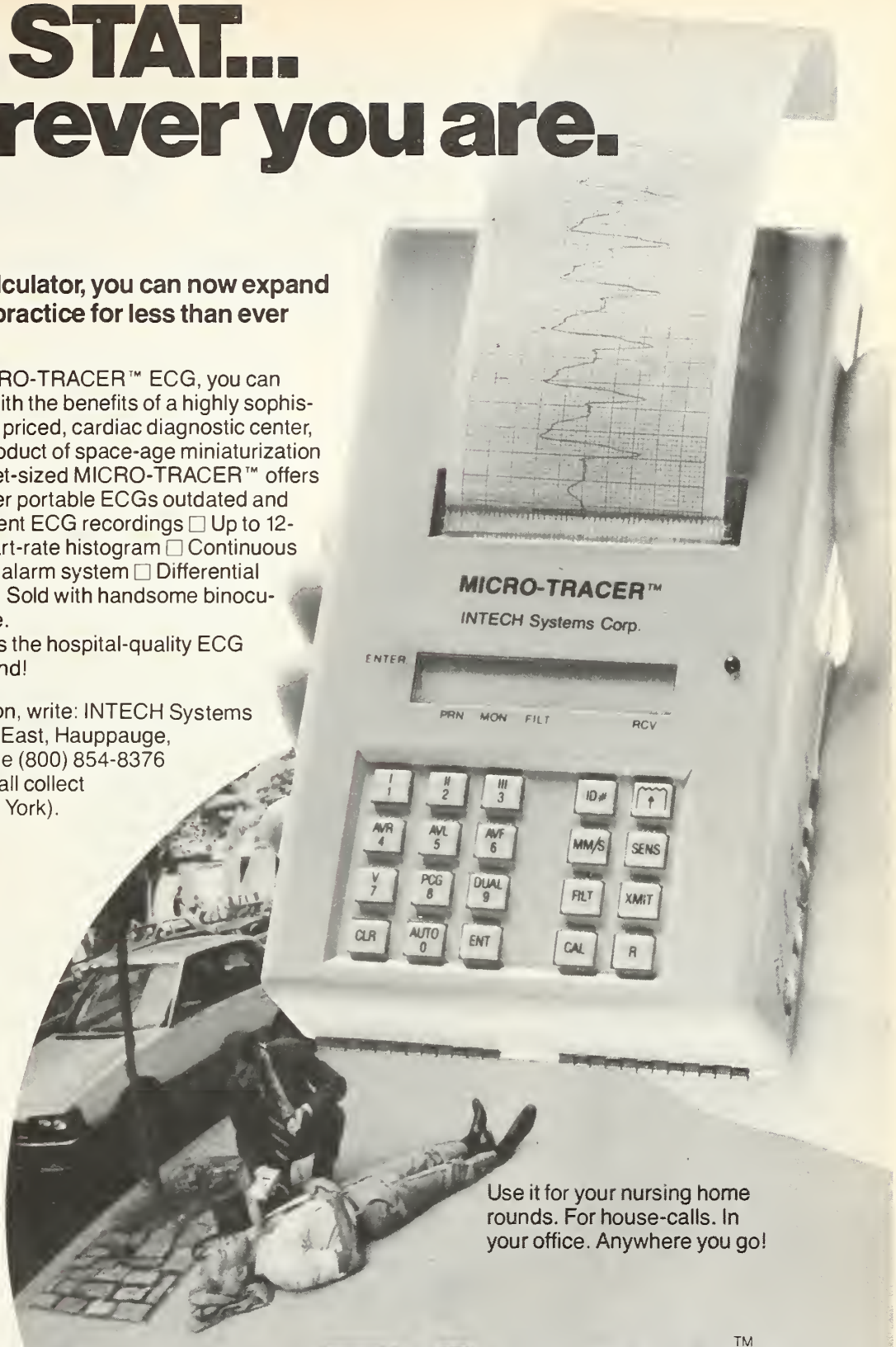

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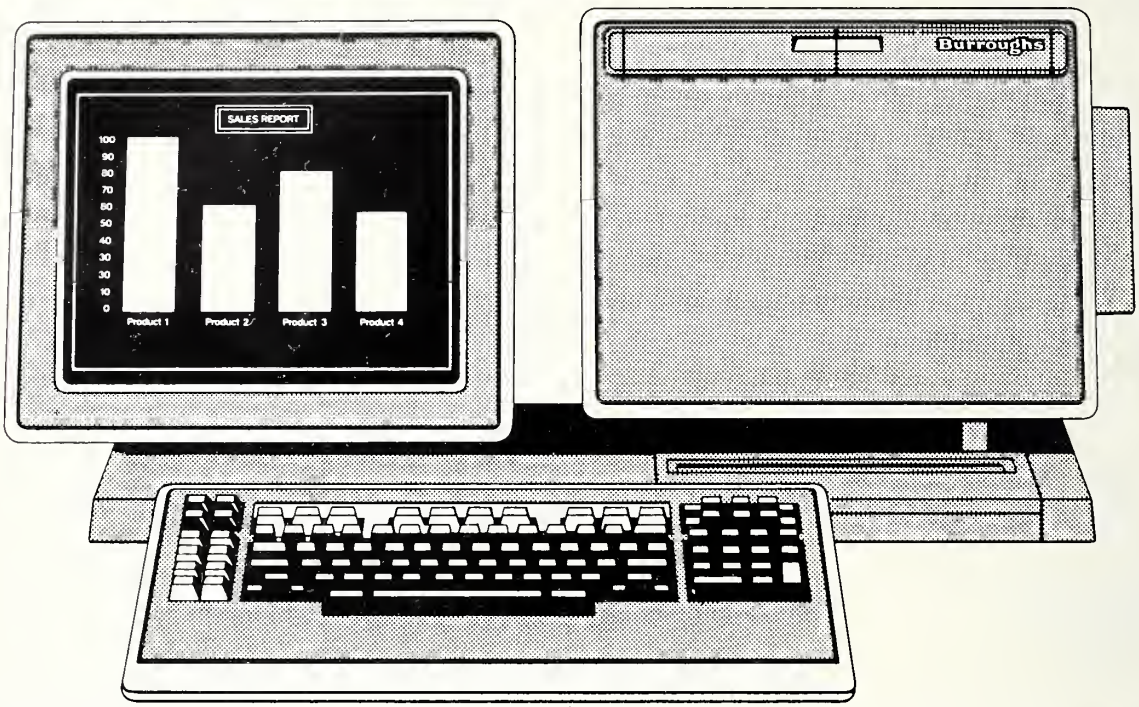

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Birth Certificate Data: How Reliable?

For over 22 years the University of South Dakota Cranio-Facial Anomalies Clinic has provided treatment coordination throughout the state. The program's primary thrust is to deliver multidisciplinary service to those needing care, at minimal cost. To this end, the program has been eminently successful. However, because primary emphasis is upon service, and funding has been limited, no attempt has been made to evaluate the results of the program in depth.

In 1984, the findings from services provided to individuals with cranio-facial anomalies (CFA) in South Dakota by the Comprehensive Health Care Services (CCHCS) and the USD-CFA Clinic have been computerized. Information utilized for the data base was from three sources: (1) currently listed patients in the CCHCS program, (2) Patients enrolled at the USD-CFA Clinic, and (3) J. B. Gregg(s), M.D. medical office files. These sources provided a broad data base relating to well over 800 patients.

While data were being processed, very interesting and relevant information was forthcoming. This will be reported later. However, unexpected findings appeared, findings that should be important to health care professionals in South Dakota.

After assembly of the primary data base, to locate CFA patients who had "fallen through the cracks," and whose names were not in the available sources, birth certificates in the Health Department's computerized Vital Statistics Division were scanned for those coded 749.0 (cleft palate), 749.1 (cleft lip), and 749.2 (cleft palate and lip), for a sample interval, 1980, 1981, 1982, and 1983 (51,341 life births; 1286 reported anomalies).

Twenty-eight CFA patient names appeared in the data base file but not on birth certificates. Two of these were born out of state. Sixteen CCHCS and CFA Clinic program enrollees, not listed on birth certificates, had functional anomalies that could easily have been missed at birth.

However, ten patient's birth certificates listed no congenital anomalies, when in actuality very definite and obvious anomalies were present. The unreported CFA anomalies included:

1. bila. complete lip and palate clefting — 1 case
2. unila. complete lip and palate clefting — 3 cases (one was blind, deaf, and affected by severe fetal alcohol syndrome)
3. unilateral cleft lip — 1 case
4. palate cleft, soft and hard palate — 3 cases (one also has mild external ear deformity)
5. palate cleft, soft palate only — 1 case
6. congenital macroglossia, micrognathia — 1 case (and familial history of other congenital anomalies)

The children with anomalies reportable upon birth certificates were delivered at communities throughout the state by physicians (Obstetricians = 6; OB & other specialty = 1; Family Practice = 2), and one nurse-midwife.

The obvious question is: if ten children had unreported overt craniofacial anomalies, how many other anomalies elsewhere in the body escaped documentation? An additional pungent question is; if this number of anomalies were omitted from birth certificates during only four years, how many inaccurate birth certificates were filed in other years?

To answer this question, South Dakota birth certificates for the interval 1970-1979 (116,281 live births; 2678 anomalies) were compared with the CCHCS and CFA Clinic files. Births were listed for 328 individuals during these ten years in the latter files, but for 112 (34.1%) birth certificates indicated no CFA. The unreported anomaly was obvious in 69 of these individuals (61.6%). In addition, anomalies as reported on a significant number of birth certificates were not corroborated clinically. Birth certificates reported CFA for 52 individuals (749.0 = 45, 749.1 = 6, 749.2 = 1), whose names weren't in CCHCS and CFA lists, suggesting they probably received treatment other than through the CCHCS and CFA programs.

The best way to assemble demographic information is with vital statistics. However, vital statistics are only as good as the information source. When physical defects obvious at birth are not reported upon birth certificates, inferences from the data base are questionable. The validity of birth certificates to compile vital statistics relating to cranio-facial anomalies has been scrutinized previously, and found wanting.[1]

In the past ten years health care delivery in South Dakota has changed tremendously in quality and quantity, an outstanding example being the salvage of neonates. Information from South Dakota Vital Statistics corroborates the significant progress in this facet of medicine. However, if inaccuracies appear in one portion of the state's vital statistics, the validity of other statistics may be questioned.

A chain is only as strong as its weakest link. The physician who delivers the newborn, or supervises the delivery is responsible for accurate information upon birth certificates. If reliable data are to be obtained for vital statistics, each attendant at the accouchement must be certain that information is placed upon the certificate of birth, and is accurate. If information is forgotten or is discovered later, an amended birth certificate should be filed with the Vital Statistics Division of The Health Department.

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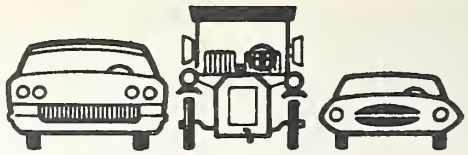

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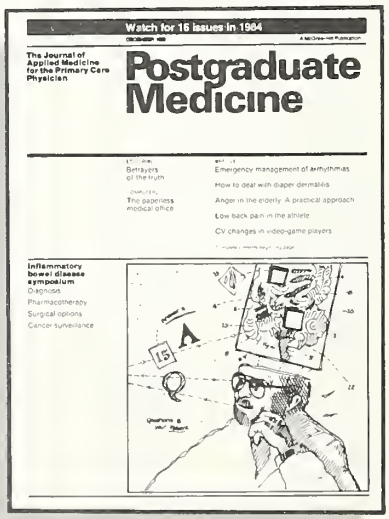
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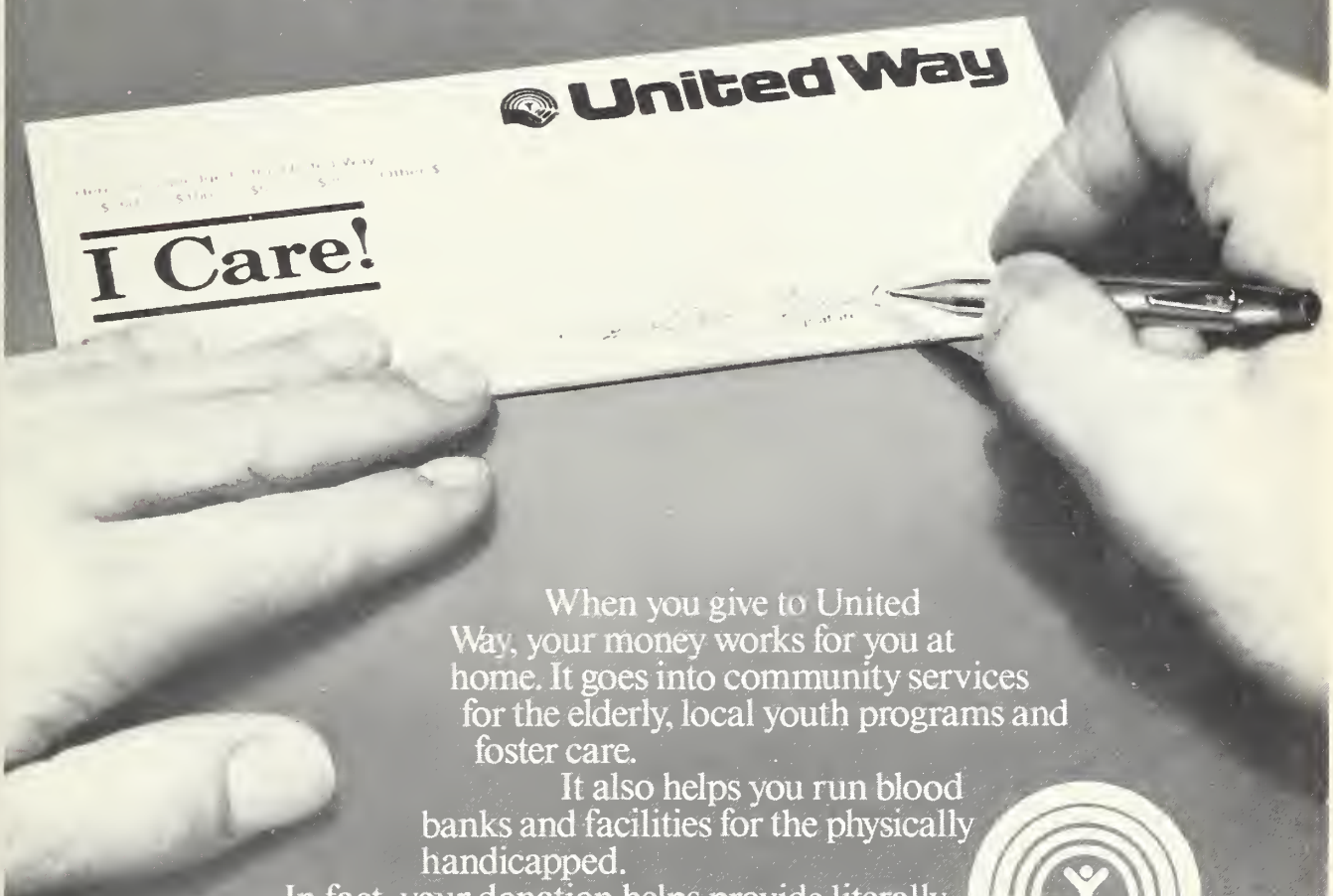

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A public service message from this magazine and the Advertising Council.

The Day All Volunteers Quit

Erma Bombeck wrote recently of a dream she had that every volunteer in the country, disillusioned with the lack of compassion, had set sail for another country.

She saw herself standing on the pier, feeling good because she thought of volunteers as a bunch of "yes" people who could have spared themselves a lot by saying "no."

Then she went home, passing quiet hospitals where there were no flowers in patients' rooms. The home for the aged was "like a tomb." The infirm sat in wheel chairs that would never move. Social agencies had closed their doors, health agencies cancelled research, alcoholics cried out in despair. She fought in her sleep to catch a glimpse of the ship again, because "it was to be my last glimpse of civilization . . . as we were meant to be."

We don't want this to happen. And we don't think you do. Why don't you join us?

WE NEED YOU . . . to

Welcome opportunities for service
Educate people to take care of their health

Nurture good attitudes about the medical profession
Ensure the access of all Americans to good health and health care
Extend the abilities of our spouses to provide good health care
Develop new ways to help our communities
Yield to the desire to be of service
Offer more needed programs
Utilize all available resources

WE NEED YOU to do all these things.

"Citius, altius, fortius." The Latin motto for the Olympic Games: "Swifter, higher, stronger." The flame is extinguished; the pins are traded; the traffic's back to dismal, and Fort Knox, again, is the nation's principal repository of gold. So what is left? Of course, the **Auxiliary Parade of Champions!**

The challenge of auxiliary functions has begun to echo across the districts. You have pledged your support and enthusiasm for an active 1984-1985. Thanks for continuing to share your auxiliary pride and enthusiasm. We really can do more together!

Mary Wrage

Mary Wrage, President
South Dakota State Medical Association Auxiliary

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The King and Queen of this year's Nordland Fest, held annually in Sioux Falls, are both members of the S.D. State Medical Association, **Eileen Devick,** an auxiliary member and **Dr. Richard Friess,** a physician member.

Mrs. Devick has contributed time and energy to almost every fest since it began. She has been a member of the board of directors; a member of the Norland Heritage Foundation board and is president of the Nordland Heritage Guild.

Dr. Friess, a family practice physician in Sioux Falls, was selected as king because of his role in expanding the Nordland Fest into a citywide celebration as president of the Sioux Falls Chamber of Commerce.

* * * *

The American College of Physicians announced that **William Taylor, M.D.** of Aberdeen has assumed office as a Governor of the 57,000 member national medical specialty society. Dr. Taylor will hold his post as the ACP Governor of South Dakota for four years.

Dr. Taylor is a specialist in internal medicine and is a clinical professor of medicine at the USD School of Medicine.

* * * *

Robert Bloemendaal, M.D., Rapid City and **Roscoe Dean, M.D.,** Wessington Springs, were re-elected to the board of directors of South Dakota Blue Shield. Other physicians on the board include: **Drs. J. O. Mabee,** Mitchell; **Winston Odland,** Aberdeen; **Dennis G. Ortmeier,** Sioux Falls; **John Stransky,** Watertown; and **Robert E. Van Demark,** Sioux Falls. Dennis Ortmeier was re-elected chairman of the board and Roscoe Dean was re-elected vice chairman.

Dr. James S. Pucelik, orthopedic surgeon, has begun his practice in Deadwood. He was born in Omaha, Neb. He received his M.D. degree in 1965 from the Univ. of Oregon Medical School in Eugene, Oregon. He completed a three year residency at San Bernardino, California County Hospital and a three year orthopedic residency at St. Francis Hospital in Wichita, Kans. He also served two years in the U.S. Air Force.

Dr. Pucelik and his wife Sharon came to the Black Hills from Yuma, Ariz., where he practiced the past eleven years. They have three grown children.

* * * *

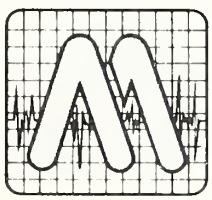
Charles Warrender, M.D. has opened his psychiatry practice in Pierre. A native of Kansas, Dr. Warrender came to South Dakota from Joplin, Mo. where he had been in private practice since August 1983. The previous eight years had been spent in the Air Force.

Dr. Warrender received his M.D. degree from the Univ. of Kansas in 1960. He did a rotating internship at William Beaumont Army Hospital, El Paso, Texas; a two year psychiatry residency at Letterman General Hospital, San Francisco, Calif. and a one year residency at Baylor Univ. Medical Center in Houston, Texas. He received his master of public health from Tulane Univ. in New Orleans in 1980; and did a public health residency in 1980-81 at the Hayes Army Medical Center in Monterrey, Calif.

Dr. Warrender and his wife Laura have three grown daughters and three grandchildren.

* * * *

Aberdeen family physician, **Dr. Karlis Zvejnieks,** completed continuing education requirements to retain active membership in the American Academy of Family Physicians.



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Edward Maloney, M.D. has recently opened his family practice in Custer. He is a native of Washington and he came to South Dakota from Deer Park, Wash. where he was in private practice.

Dr. Maloney received his M.D. degree from the Univ. of Missouri at Columbia in 1976. He completed his internship at Deaconess Hospital, Spokane, Wash. and his residency at Yakima Valley in Yakima, Wash. Following his training, Dr. Maloney practiced for two years in Montana and four years at Deer Park where he has been an associate clinical professor in family medicine at the Univ. of Washington.

Dr. Maloney and his wife, Marcia, have five sons.

* * * *

Richard P. Holm, M.D., internal medicine, Brookings; and **David Elson, M.D.,** medical oncology, Sioux Falls, have both been notified that they have been elected to Fellowship in the American College of Physicians.

* * * *

Dr. Robert Rietz, ear, nose and throat specialist in Brookings, has passed the certifying exam of the American Board of Otolaryngology.

Colonel Eldon E. Bell of Webster has been selected to attend the U.S. Army War College resident course at Carlisle Barracks, Pennsylvania. Dr. Bell is one of ten Army National Guard officers who will represent the entire Army National Guard in a course of instruction that is attended primarily by active component officers, many of whom are destined to be the future leaders of our nation's defense forces.

* * * *

Ila Lushbough, Brookings, has been nominated as an AMA Auxiliary North Central Director; and **Marie Hovland,** Aberdeen, has been appointed to the AMA Auxiliary Committee on Legislation. Both Mrs. Lushbough and Mrs. Hovland are past presidents of the South Dakota State Medical Association Auxiliary.

* * * *

The USD School of Medicine's Department of Family Medicine has recently awarded the Edward J. Batt Memorial Award to **Dr. R. Curtis Jahraus, M.D.** Dr. Jahraus is a family physician in Pierre and also a clinical professor of family medicine at the USD School of Medicine.

The Batt Memorial Award was established in memory of the late Dr. Edward Batt and is given annually to an outstanding faculty member (full-time or clinical) of the Department of Family Medicine.

South Dakota Society Of Pathologists

Officers for 1983-84

Jerry L. Simmons, M.D., President
John F. Barlow, M.D., Vice President
Beth L. Johnson, M.D., Secretary-Treasurer

Dr. Barbara K. Spears, family practice physician in Pierre, was named the 1984 "Woman of Achievement" by the Pierre-Fort Pierre Chapter of Business and Professional Women. The club annually honors a woman who has excelled in her profession and in service to the community.

* * * *

B. R. Skogmo, M.D., family practice physician in Mitchell, has retired after practicing for 36 years in that community. He is a native of South Dakota. Dr. Skogmo received his medical degree at the Univ. of Minnesota in 1940 and interned for one year at Columbia Hospital in Milwaukee, Wisc. During World War II, he served as a first lieutenant and later as a captain in the U.S. Army, from 1941-45.

In 1951, Dr. Skogmo built the first medical clinic in Mitchell, where he has practiced since. He has always been affiliated with both local hospitals and has served as chief of staff at each hospital. He is a charter member of the AAFP and a member of the South Dakota State Medical Association and the AMA.

* * * *

Dr. Tony Javurek, emergency/trauma physician in Sioux Falls, has become certified in emergency medicine. He also is certified in family practice.

* * * *

Mark Hendricks, M.D., a native of Minnesota, has recently begun practice in Huron. Dr. Hendricks specializes in internal medicine. He received his medical degree from the University of Minnesota and served a three year residency in LaCrosse, Wisc.

Dr. Hendricks and his wife, Jill, have two sons.

* * * *

Dr. John Richey has moved to Britton to begin his family practice there. Dr. Richey came to South Dakota from Milwaukee, Wisc. where he has just completed his family practice residency. He received his medical degree from the University of Vermont College of Medicine in 1981.

* * * *

Dr. Edwin Gerrish, Watertown, has received notification that he has been named a diplomat of the American Board of Surgery. Dr. Gerrish is a specialist in general and vascular surgery.

* * * *

A Watertown physician, **Dr. Linda Peterson,** was selected as Woman of the Year by the local Beta Sigma Phi sorority chapters. The award is presented to a woman in the community, who has made an outstanding contribution in her field, during the past year.

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ENDOWMENT ASSOCIATION — ALUMNI FOUNDATION

Often times there are questions about differences between the South Dakota Medical School Endowment Association and the USD School of Medicine Alumni Foundation. Both were founded for the benefit of the Medical School and its students; however, each has its own distinct goals and objectives. Hopefully the following will clarify the difference in activities between these two organizations.

ENDOWMENT ASSOCIATION

1. Primary activity is provision of loans to USDSM students.

ALUMNI FOUNDATION

1. Supports activities and projects recommended by the administration and faculty of USDSM.
2. Supports USDSM Alumni activities and services.
3. Supports USDSM student activities and services.

Additional information can be obtained by contacting:

South Dakota Medical School
Endowment Association
608 West Avenue, North
Sioux Falls, SD 57104
(605) 336-1965

School of Medicine Alumni
Foundation
USDSM
Vermillion, SD 57069
(605) 677-5498

Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Friday, September 14, 1984, in Sioux Falls, South Dakota. Following are items of business transacted at this meeting.

1. LOCUM TENENS PROGRAM. The Council directed the executive office to survey the membership regarding the need for an Association sponsored locum tenens physician program in South Dakota and the availability of physicians to work as locum tenens. The Commission on Medical Service will study the results of this survey and make a recommendation to the Council.
2. PUBLIC RELATIONS. Educating the membership in the area of public relations will be a major emphasis of the Association according to Council action. Also, the Association should develop information for patients on timely issues, disseminating that through the media via district medical societies or individual physicians. The Association will obtain the advice of specialists in the Division of Public Relations at the AMA and a special session with these resource people and the members of the Commission on Internal Affairs, Communications and Liaison will be held.
3. NURSING EDUCATION. Council action supports improved quality of nursing education and training in South Dakota but opposes the requirement of a Baccalaureate Degree.
4. LOBBYIST FOR STATE MEDICAL ASSOCIATION AND SOUTH DAKOTA CHAPTER, AMERICAN ACADEMY OF FAMILY PHYSICIANS. The Council considered a proposal from the South Dakota Chapter, AAFP to hire a joint lobbyist. Concern was expressed by Council members that this could cause fragmentation within the State Association, and therefore action was taken to have lobbying efforts continue as they have.
5. HONORARY LIFE MEMBERS. Four doctors were elected to honorary life membership in the State Medical Association. They are: Hollis Ahrlin, M.D., Rapid City; Mary Sanders, M.D., Redfield; Warren Anderson, M.D., Sioux Falls; and Valdis Brakss, M.D., Watertown.
6. MEDICARE. Lengthy discussion was held regarding the Deficit Reduction Act and the effects the Medicare amendments have on the doctor-patient relationship. The Council directed that the executive office take an aggressive approach with a public information program which may include dissemination of information through any aspect of the media that may seem appropriate. ■

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Reuben J. Bareis, M.D.
Medical Director
255 Texas Street
Rapid City, SD 57701

Future Meetings

January

Diagnostic Radiology Seminars, Camino Real, Ixtapa, Mexico, Jan. 14-18. Fee: $425. 32 hrs. AMA Category I credits. Contact: Robert C. Brasch, M.D., Asso. Prof. of Rad., U. of Calif. School of Med., San Francisco, CA.

American Society for Reconstructive Microsurgery, MGM Grand Hotel, Las Vegas, NV, Jan. 17-19. Fee: $350. Contact: Berish Strauch, M.D., Pres., Am. Soc. for Reconstructive Microsurgery, 3331 Bainbridge Ave., Bronx, NY 10467. Phone: (212) 920-5551.

Eighth Annual San Diego Postgraduate Assembly in Surgery, Sheraton Harbor Island, San Diego, CA, Jan. 21-25. 28 hrs. AMA Category I credits. Contact: Off. of CME, M-017, UC San Diego School of Med., La Jolla, CA 92093. Phone: (619) 452-3940.

National Thermography Seminars, Houston, TX, Jan. 24-26. Contact: Paula Hobbins, Seminar Cood., Thermal Image Analysis, Inc., 5510 Medical Circle, #B, Madison, WI 53719. Phone: (608) 273-0362.

Trauma Management 1985, Vacation Village Resort, San Diego, CA, Jan. 28-30. 20 hrs. AMA Category I credits. Contact: Off. of CME, M-017, UC San Diego School of Med., La Jolla, CA 92093. Phone: (619) 452-3940.

February

Clinical Topics in Internal Medicine, Snowbird resort, Snowbird, Utah, Feb. 2-9. Contact: Off. of CME, Scott and White, 2401 S. 31st St., Temple, TX 76508. Phone: (817) 774-2350.

Coping with Loss: The Experience of Grief, Seely Conf. Cntr., Menninger Foundation, Topeka, KS, Feb. 6-8. Fee: $185. 13 hrs. AMA Category I credits. Contact: Jayne Roberts, Div. of CME, The Menninger Found., Box 829, Topeka, KS 66601. Phone: (913) 273-7500, ext. 5992.

Nineteenth Annual Symposium on Cosmetic Surgery, Hyatt Regency, Miami, FL, Feb. 7-9. Fee: $600. CME credit hours available. Contact: Thelma MacGregor, Cedars Med. Cntr., 1400 N.W. 12th Ave., Miami, FL 33136.

March

Review and Update: General Pediatrics and Family Practice, Sheraton Hotel, Cancun, Mexico, Mar. 3-8. Fee: $400. 24 hrs. AMA Category I credits. Contact: Marge Adey, Cntr. for CME, U. of Neb. Med. Cntr., 42nd and Dewey Ave., Omaha, NE 68105. Phone: (402) 559-4152.

Common Problems in Pediatrics VII, Little America, Salt Lake City, Utah, Mar. 6-8. AMA Category I & AAFP credit hours available. Contact: Wendy Ellis, 175 N St., Salt Lake City, UT 84103. Phone: (801) 363-1887.

Brain Insults in Infants and Children: Pathology, Evaluation, Diagnosis and Acute Management. Holiday Inn, Embarcadero, San Diego, CA, Mar. 7-9. 17 hrs. AMA Category I credits. Contact: Off. of CME, M-017, UC San Diego School of Med. La Jolla, CA 92093. Phone: (619) 452-3940.

Clinical Topics in Gynecologic Endocrinology for the Primary Care Physician, Prospector Square Resort, Park City, Utah, Mar. 9-16. Contact: Off. of CME, Scott and White, 2401 S. 31st St., Temple, TX 76508. Phone: (817) 774-2350.

Sixth Annual Mammoth Mountain Emergency Medicine Ski Conference, Mammoth Lakes, CA, Mar. 10-15. Fee: $325. 20 hrs. AMA Category I & AAFP credits. Contact: Daniel Abbott, M.D., Prog. Dir., Medical Conf., Inc., P. O. Box 52-B, Newport Beach, CA 92662. Phone: (714) 650-4156.

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South Dakota Journal of Medicine	0 0 3 8 3 3 1 7	9/28/84
3. FREQUENCY OF ISSUE	3A. NO. OF ISSUES PUBLISHED ANNUALLY	3B. ANNUAL SUBSCRIPTION PRICE
monthly	12	10.00

4. COMPLETE MAILING ADDRESS OF KNOWN OFFICE OF PUBLICATION *(Street, City, County, State and ZIP Code) (Not printers)*
608 West Avenue, Sioux Falls, SD 57104

5. COMPLETE MAILING ADDRESS OF THE HEADQUARTERS OF GENERAL BUSINESS OFFICES OF THE PUBLISHER *(Not printer)*
608 West Avenue, North Sioux Falls, SD 57104

6. FULL NAMES AND COMPLETE MAILING ADDRESS OF PUBLISHER, EDITOR, AND MANAGING EDITOR *(This item MUST NOT be blank)*

PUBLISHER *(Name and Complete Mailing Address)*
South Dakota State Medical Association,
608 West Avenue, North, Sioux Falls, SD 57104

EDITOR *(Name and Complete Mailing Address)*
R. E. Van Demark, M.D. 608 West Avenue, North, Sioux Falls, SD 57104

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Volume XXXVII December 1984 Number 12

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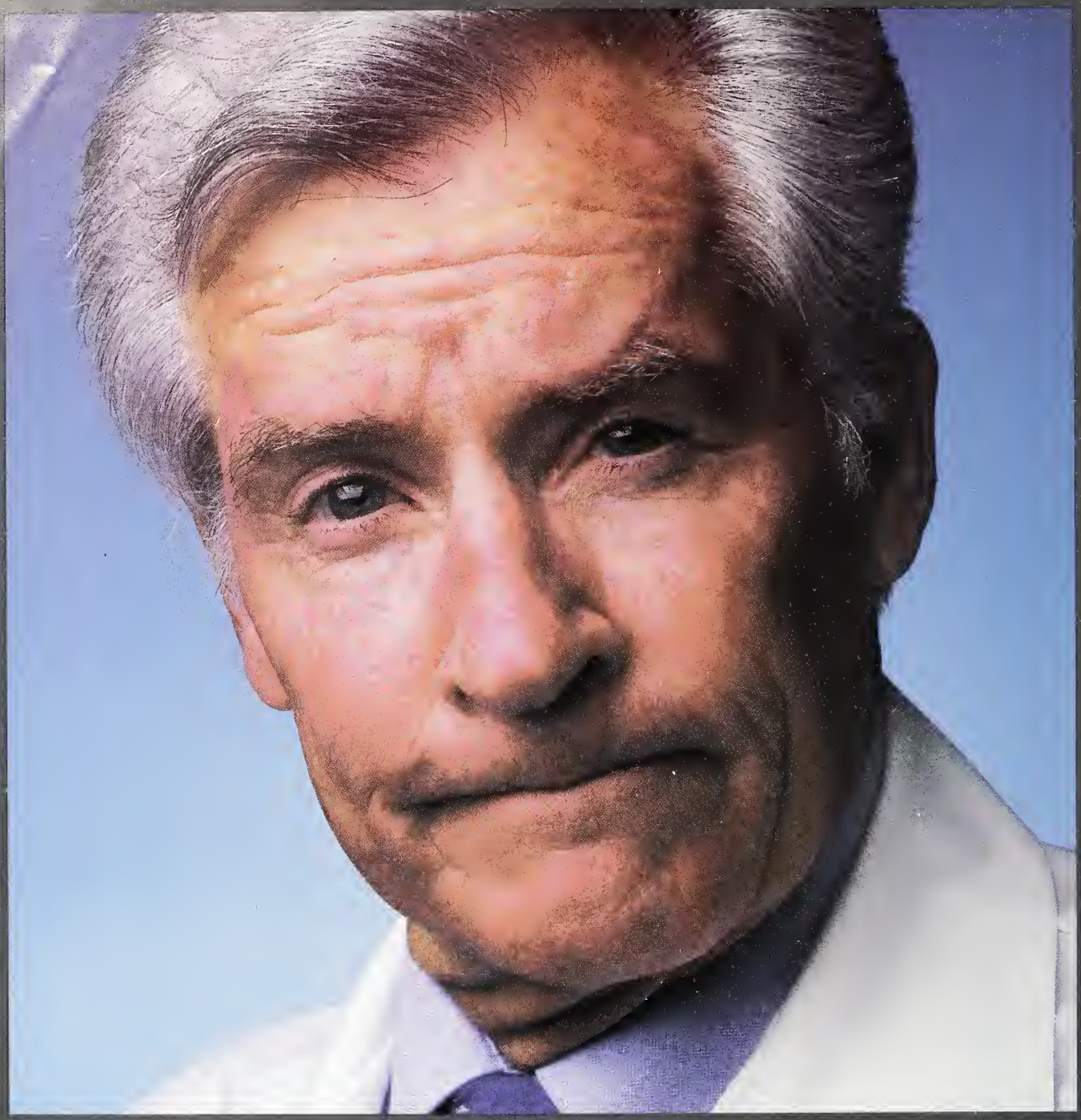

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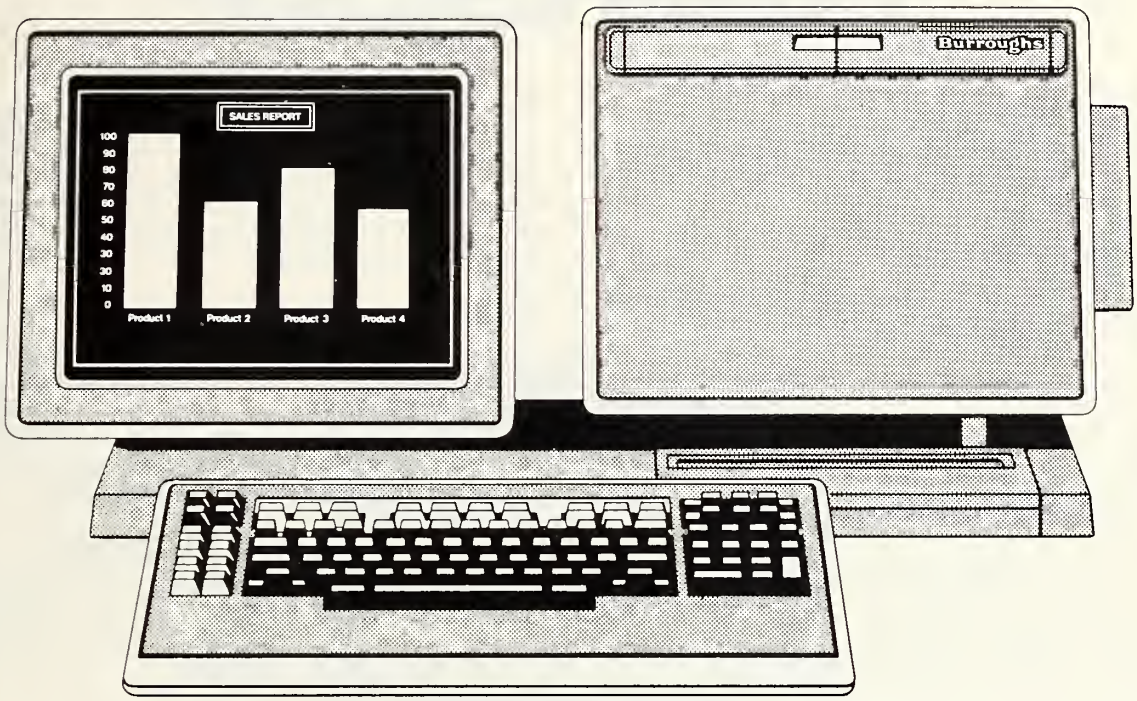

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SOUTH DAKOTA JOURNAL OF MEDICINE

Volume XXXVII/No. 12 December 1984 (ISSN 0038-3317)

Official Publication of the
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Medical Association

the South Dakota Chapter
of The American Academy
of Family Physicians

the South Dakota Chapter
of the American College
of Surgeons

the South Dakota
Psychiatric Association
of the American
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SOUTH DAKOTA JOURNAL OF MEDICINE (ISSN 0038-3317) is published monthly for $10 per year domestic, $15 per year foreign, $1.50 for single copy, by South Dakota State Medical Association, 608 West Avenue, North, Sioux Falls, SD 57104. Second class postage paid at Sioux Falls, South Dakota 57101 and additional mailing offices. POSTMASTER: Send address changes to SOUTH DAKOTA JOURNAL OF MEDICINE, 608 West Avenue, North, Sioux Falls, SD 57104.

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and best wishes for the New Year

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Lorin Pankratz, Susan Jahraus

SOUTH DAKOTA STATE MEDICAL ASSOCIATION

Twenty-Eight Year Old Caucasian Female With Left Flank Pain and Renal Mass

Eric Ruhe, M.D.*
John A. Ochsner, M.D.†
Discussers

John F. Barlow, M.D.‡
Editor

Case #1037473 (83-9710)

This 28 year old single Caucasian female was admitted to Sioux Valley Hospital because of the recent onset of left flank pain.

The patient had had prepubertal bladder problems of nonspecific nature. She had been hospitalized for evaluation and no abnormality was noted. She had had no problems with the genitourinary tract in recent years and voiding was normal. There were no other significant symptoms on review of systems. Her only other serious illnesses had been episodes of pain in her knees and calves with swelling and tenderness diagnosed as phlebitis four years and two years prior to admission. These were both treated conservatively with rest, elevation and compressive stockings as well as antiinflammatory drugs. Eleven months prior to admission, the patient had a third episode of calf pain, tenderness and swelling. She was anticoagulated and the symptoms and signs disappeared. Fifteen days prior to admission she developed an episode of acute left flank pain without radiation and one day later developed dark gross hematuria. At that time the prothrombin time was 40 seconds with a control of 13 seconds and coumarin medication was discontinued. She was hospitalized for several days and noted to have a low grade fever for which she was treated with antibiotics. An intravenous pyelogram showed a left lower pole renal mass with moderate dilatation of the upper pole collecting system. The hematuria cleared but she continued to have dull left flank pain and low grade fever. An ultrasound study also showed left hydronephrosis. She was referred for further evaluation. She had no previous history of hospitalizations except for an appendectomy ten years prior to admission.

PHYSICAL EXAMINATION: Obese young woman in no distress. Pulse 88/minute and regular; respirations 16/minute and regular; blood pressure 130 systolic and 78 diastolic; temperature 37.5°C. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. There were no masses in the breasts. The heart had a regular sinus rhythm without murmurs. The abdomen was obese but there were no palpable organs, masses, spasm or tenderness. The left flank area showed some slight tenderness on percussion. Pelvic examination was within normal limits. Examination of the extremities showed no evidence of swelling, tenderness or heat. The pulses were unremarkable. Neurologic examination was within limits.

LABORATORY DATA: Urinalysis — yellow, cloudy; specific gravity 1.030, pH 5.0, negative for glucose, ketones, bilirubin and protein; hemoglobin 3+; sediment 20-25 leukocytes per high power field and 10-15 red cells per high power field and with many squamous epithelial cells and amorphous urates. Admission hemoglobin 9.7 gm/dl, hematocrit 30 Vol/dl with normal red cell indices, total leukocyte count 8.8 x 10^9/L with a differential count of 57% segmented neutrophils, 5% neutrophilic bands, 23% normal lymphocytes, 8% monocytes, 6% eosinophils and 1% basophils. The platelet count was 444 x 10^9/L. Erythrocyte sedimentation rate was 121 mm/hr; plasma electrolytes — sodium 137 meq/l, potassium 3.5 meq/l, chloride 105 meq/l and CO_2 content 25 mm/l. Lactic dehydrogenase (LDH, LD), alkaline phosphatase, aspartate aminotransferase (SGOT, AST), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid were within normal limits. Cholesterol was 147 mg/dl. A rheumatoid arthritis test was negative. A urine culture showed no growth after 48 hours.

Chest x-ray showed minor discoid atelectasis at the left base. There was no other abnormality. Review of outside intravenous pyelogram revealed a large left kidney mass. A computer tomogram (CT) of the abdomen and pelvis showed a large left renal mass consistent with hypernephroma (Fig 1). A venous doppler and plethysmography for thrombophlebitis were negative. An operation was performed.

* Resident in Family and Community Medicine, program affiliated with USD School of Medicine, Sioux Falls, SD.
† Urologist Sioux Valley Hospital and Central Plains Clinic. Faculty, USD School of Medicine, Sioux Falls, SD.
‡ Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital. Professor of Pathology, USD School of Medicine, Sioux Falls, SD.

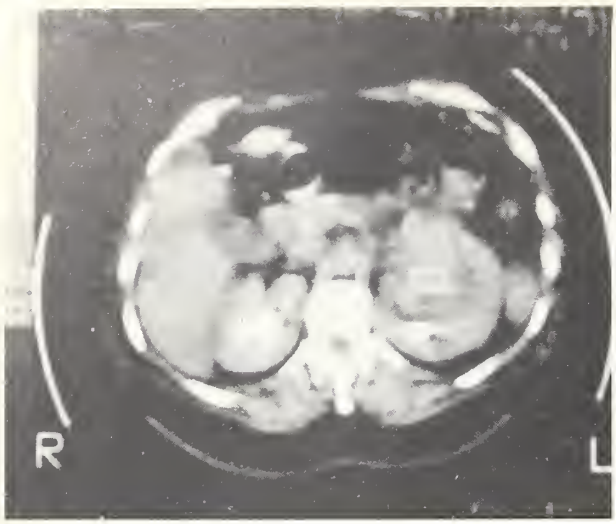

Figure 1
Central mass in left kidney (square) with lucent line demarcating the lesion.

DR. RUHE: In this case, the results of the CT scan and the subsequent data presented are "consistent with hypernephroma." Since a renal mass had been identified as the principle pathology, it would seem appropriate to outline renal tumors and present a differential diagnosis which contains entities which (even though quite unlikely) could cause renal masses.

I would begin the discussion by ruling out any extrarenal causes of abdominal masses as a source of metastatic disease to the kidney by calling attention to the objective evidence from physical and laboratory examination (i.e. no hepatomegaly, splenomegaly, normal pelvic examination, no weight loss or change in bowel habits, hematochezia, melena, etc). To be more specific, it would seem unlikely that we have to look further than the renal parenchyma since the intravenous pyelogram (IVP) demonstrates a "left lower renal pole mass" and an ultrasound and CT scan confirm this finding. These diagnostic modalities would also rule out cystic disease of the kidney and an unremarkable white count and only low grade fever tend to exclude a renal abscess.

BENIGN RENAL NEOPLASMS

1. **Cortical adenoma:** A rather common entity that is said to be found in 7-20% autopsies. The origin is the renal tubules giving rise to a tumor of papillary structure lined by regular cuboidal cells or one composed of clear cells within the renal cortex. They are more common in males (4:1) and occur in the 40-50 year old age group. They are grossly pale yellow and usually less than 2.0 cm. in greatest diameter. Although considered benign, their size seems to be the most salient factor in their propensity to become malignant. If they are found to be greater than 3 cm., it becomes difficult to differentiate histologically large adenomas from small renal cell carcinomas. It has also been postulated that these adenomas are the "subsoil" for subsequent renal cell carcinoma. They may rarely produce hematuria secondary to their ability to erode blood vessels, or present as a filling defect on IVP by distortion of calyces or renal pelvis. They also rarely can produce pain by the tumor pressure on renal structures and nerves.
2. **Renal medullary fibroma (Hamartoma):** These are usually gray-white firm tumors found within the pyramids of the kidney and less than 0.1 cm. in diameter composed of mature well differentiated fibroblasts and with no malignant potential. Although not malignant, they can occasionally become large enough to create a mass effect with subsequent abdominal pain and pressure deformity on the collecting system detectable on the IVP.
3. **Angiomyolipoma:** These are hamartomatous malformations consisting of a mixture of vessels, smooth muscle and fat as the name clearly implies. This is a common entity found in tuberous sclerosis (Bourneville's disease) which consists of mental retardation, epilepsy, and sebaceous adenoma of the skin of the face. In this hereditary disorder, angiomyolipoma is found in over 80% of cases. It is frequently bilateral, has a predilection for women and has an onset after 40 years. This lesion too can cause recurrent painless hematuria and abdominal pain. Arteriogram demonstrates multiple small arterial aneurysms and a diagnostic pattern may be seen on CT. In this particular case angiomyolipoma is an unlikely consideration because it does not satisfy many of the aforementioned criteria for a definitive diagnosis on the CT. It should be noted that the lesion can occur in the absence of tuberous sclerosis so we cannot completely exclude this lesion.

   Other miscellaneous benign tumors should be considered such as renin producing juxtaglomerular cell tumor (hemangiopericytoma), neurofibroma, rhabdomyoma (very rare) and a dermoid cyst of the kidney.
4. **Endometriosis:** A rather unusual disease entity that could be considered with a history of abdominal pain, hematuria and abnormal IVP is renal involvement by endometriosis. This has been described as a hemorrhagic gray mass in the kidney and can be demonstrated on IVP as an enlarged renal outline with deformities of the renal pelvis and calyceal system. Endometriosis

is unlikely in this particular case with a normal pelvic examination and no history of dysmenorrhea.

MALIGNANT RENAL TUMORS

1. **Nephroblastoma (Wilm's tumor):** This is the most common abdominal tumor in the less than 10 year age group. Sixty-five percent are found before 3 years of age and 75% before 5 years of age. Palpation of a large unilateral mass is a common presentation but 7-10% are bilateral. Wilm's tumor can be extremely aggressive, rupture through the renal capsule, invade perirenal tissue and even bowel mesentery. A predilection for pulmonary metastases which, at times, are present at the primary diagnosis is a notable feature. Good therapeutic results have been reported if early diagnosis is made and prudent implementation of chemotherapy, radiotherapy and surgery are employed.

   In adults (over 18 years) prognosis is not as optimistic. Although extremely rare (only 190 reported cases) many of the symptoms and laboratory findings of renal cell carcinoma are present in Wilm's tumor. Due to the fact that it is such a rare tumor in adults, it is probably not a strong possibility in this case.
2. **Hypernephroma or renal cell carcinoma:** The name "hypernephroma" is derived from the tumor's resemblance to "clear cells" of the adrenal cortex. Subsequently, it was thought to be derived from adrenal rest cells in the kidney. Actually these neoplasms arise from tubular epithelium. Therefore, it is a renal adenocarcinoma. They constitute 1-3% of all visceral carcinomas, 85-90% of renal cell carcinomas occur in older individuals (6th-7th decade) with a 3 to 1 male predominance. Morphologically they are a bright yellow to gray-white, solitary and usually unilateral. They more commonly occur in the upper poles as spherical masses (3-15 cm). Their growth is considered "expansile" in that they compress renal parenchyma rather than infiltrating it. The tumor may also bulge into calyces and the renal pelvis or even extend into the ureter. Over 50% of renal cell carcinomas invade the renal vein as a solid mass of cells and further extension into the inferior vena cava is not infrequent.

Clinically, the "not so classic triad" of renal cell carcinoma has been described i.e. costovertebral angle tenderness, palpable mass and hematuria. Only 10% of cases present with all three criteria. Hematuria alone is seen in 90% of cases. In three percent of the patients, fever is the only initial presenting symptom. This is thought to be secondary to the endogenous release of pyrogens. Weight loss, malaise, nausea, vomiting, anorexia and constipation are frequent findings.

Laboratory findings demonstrate normochromic, normocytic anemia in 50% of the cases. Erythrocytosis is seen in 5% of cases. This is felt to be secondary to erythropoietic production from the tumor or from compressed surrounding renal parenchyma. Eosinophilia, leukemoid reaction, thrombocytosis and elevated erythrocyte sedimentation rate are common. There may be abnormal liver function tests which are often reversible and do not represent hepatic metastatic disease.

This neoplasm is known as one of the great masqueraders in medicine because of multisystem involvement. It may cause hypercalcemia by metastatic bone disease or secretion of prostaglandin or parathormone like substances may produce hypercalcemia in the absence of bone disease. It may cause galactorrhea secondary to prolactin secretion or hypertension due to renin production, feminization or masculinization secondary to gonadotropic effect and Cushing's syndrome secondary to glucocorticoid production.

As mentioned earlier, renal cell carcinomas are notorious for renal vein invasion and extension into the inferior vena cava. In a study from the Mayo Clinic (1950-1973) of primary vena cava thrombosis and obstruction, carcinoma of the kidney (usually hypernephroma) clearly outnumbered carcinoma of the pancreas as a cause of inferior vena cava thrombosis.

There was no reported palpable mass in this case, but she does demonstrate left flank pain and hematuria. The patient does have a demonstrable left lower pole mass by IVP, abdominal CT scan and sonogram. There is distortion of the calyces and collecting system on the left. The patient also has a normochromic, normocytic anemia, slight thrombocytosis, and eosinophilia as well as an increased erythrocyte sedimentation rate. The patient has had a persistent low grade fever and although she does not present with hypertension, the literature suggests that only 25% of the patients with hypernephroma will have hypertension.

The episodes of thrombophlebitis could be an unrelated phenomena, but one would have to consider renal cell carcinoma as a distinct possibility since predisposition for thrombophlebitis occurs in renal cell carcinoma. It is of interest to note that 15 days prior to admission, the patient had been on coumadin. She developed acute flank pain and gross hematuria, and the coumadin was discontinued. Although speculative, the hematuria and pain could have been prompted by hemorrhage into a renal cell carcinoma which is noted to be a highly vascular and invasive

tumor.

Another differential diagnosis one could consider to satisfy this unusual presentation of symptoms is collagen vascular disease. This does not explain the multiple history of thrombophlebitis which could either have been an actual phlebitis or a spurious finding with the myalgia and arthralgia misinterpreted as being phlebitic in origin.

If the signs and symptoms of thrombophlebitis were indeed substantiated and treated with coumadin, then one could consider a hematoma of the kidney. I have reviewed the radiologic literature which indeed mentions hematomas more commonly in the lower poles, but an extensive medical literature review was not fruitful in substantiating a hematoma with this particular case presentation.

Dr. Ruhe's Diagnosis
Renal Cell Carcinoma
(Hypernephroma)

DR. BARLOW: We received a 700 gram kidney with peripelvic fat measuring 18 x 12 x 10 cm. The capsule stripped with ease and on section there was a central multiloculated red-brown mass (Fig. 2) which involved the peripelvic and pericalyceal collecting system. Microscopic examination revealed an organizing hematoma. Multiple sections were taken to exclude a renal cell carcinoma which is very vascular and can undergo spontaneous hemorrhage.

Although hematuria is a very common complication of oral anticoagulant therapy, a renal mass such as this simulating a renal cell carcinoma must be quite rare as we were unable to find such a case in a search of the literature.

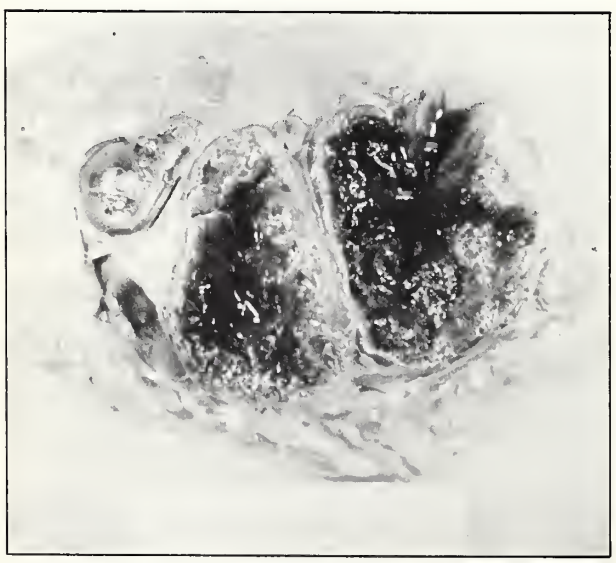

Figure 2
Central hematoma of kidney simulating neoplasm.

FINAL ANATOMIC DIAGNOSIS
CENTRAL HEMATOMA OF KIDNEY
INVOLVING COLLECTING SYSTEM

Anticoagulants are controversial but widely used agents. There is little question that many feel the use of a parenteral agent such as heparin is life saving in the prophylaxis and treatment of thrombotic disorders; but oral anticoagulants such as the coumarin derivatives, because of their ease of administration and the availability of a reproducible monitoring laboratory procedure, the prothrombin time (PT), will continue to be given commonly except during pregnancy since they are thought to be teratogenic.

However, the complications of anticoagulant therapy are not uncommon and are characterized by major and minor episodes of hemorrhage in a variety of organs. Wintzen et al.[4] described 32 intracranial and 116 extracranial bleeding episodes. Subdural, epidural, intracerebral and subarachnoid localized or diffuse bleeding occurred. In the extracranial episodes hemorrhages into skin, conjunctiva, muscle and epistaxis and hemorrhages into or from the thyroid, bronchial tree, genitourinary and gastrointestinal systems occurred. Interesting clinical problems secondary to hemorrhages in patients on anticoagulants include 1) rectus sheath hematomas which can cause abdominal pain or an abdominal mass and have been associated with shock and even death in rare cases,[2] 2) the acute onset of groin or thigh pain with a characteristic flexion and external rotation of the hip can signal serious retroperitoneal hemorrhage[3] or bilateral ureteral obstruction with renal failure may occur with a retroperitoneal bleed,[8] 3) hemorrhage into the ovary in ovulating women producing shock or abdominal pain from hemoperitoneum,[5] 4) adrenal hemorrhage with abdominal pain and sudden adrenal insufficiency,[7] 5) compressive neuropathy of various peripheral nerves.[11] 6) Skin necrosis and hemorrhage in various sites.[10]

In 1940 Link first noted hemorrhagic disease in cattle feeding on sweet clover. This led to the discovery of a variety of vitamin K antagonists, the coumarin compounds. The most commonly used in this country is warfarin or 3 alpha (acetonyl benyl) 4-hydroxycoumarin.[1]

The action of this vitamin K antagonist has been elucidated in recent years. The liver microsomes produce precursors of the anticoagulant factors II, VII, IX and X which require the gamma carboxylation of glutamates on the N-terminal fragments of the factors in order for them to achieve active binding of calcium and binding to phospholipid bilayers necessary to activate the proper sequences of the coagulation cascade. Vitamin K is vital in the gamma carboxylation step. The mechanism of warfarin

may be to competitively inhibit vitamin K at a protein receptor or transport site or it may block a process in the liver in which vitamin K is linked to an epoxide and subsequently reduced to vitamin K.[1]

The anticoagulant action of warfarin can be monitored by the highly reproducible prothrombin time. The ratio of 2.0 to 3.0 times the normal control level is commonly used; but, with the widely used rabbit thromboplastins in this country, ratios of 1.25 to 1.75 are probably more useful. A significant problem is that the test results obtained from laboratories are not identical since different thromboplastins and different instruments produce reproducible results within a single laboratory but are different from another laboratory utilizing a different thromboplastin and different instrumentation.

The vitamin K antagonists have a delayed onset of action. Although factor VII has the shortest half life and its level falls in 48 hours, optimal anticoagulation is probably not obtained for four days. Therefore, heparin is routinely used in this early period. It must be remembered that in discontinuing heparin in the switch to oral warfarin, the PT can be elevated by high levels of heparin. If heparin is administered intermittently, the plasma PT level should be drawn during a heparin trough. With continuous heparin, a decline in the PT may occur after discontinuation of heparin.

Differences in absorption, distribution and metabolism of warfarin occur in individual patients. The drug is metabolized by the liver, bound to plasma protein (97%) but not secreted in breast milk. Inherited genetic abnormalities, one an autosomal dominant, producing variation in affinity of warfarin to receptors may produce a genetic resistance or susceptibility to this drug.

Vitamin K is normally obtained by the human from two sources 1) diet and 2) production by intestinal bacteria. Many patients can develop vitamin K deficiency from decreased oral intake of vegetables. When this is combined with oral antibiotics which suppress vitamin K producing intestinal flora, marked hypersensitivity to vitamin K antagonists may develop. Vitamin K deficiency may also result from intestinal malabsorption in sprue or pancreatitis or secondary to biliary obstruction since natural vitamin K is a fat soluble vitamin and requires bile acids for absorption. The above conditions may also interfere with warfarin absorption. The effect on warfarin therapy may be hard to predict.

Liver disease can increase the sensitivity to warfarin because of depressed synthesis of coagulation factors and vitamin K metabolism. Hepatitis, cirrhosis, or even hepatic congestion secondary to congestive heart failure may have this effect. Decreased sensitivity to warfarin may occur when diuretic therapy reduces hepatic congestion. Fever and hyperthyroidism can also increase sensitivity to warfarin by increasing the utilization of vitamin K.

Most important, however, are the many drugs which can alter the effects of warfarin. Most interfere with absorption or alter metabolism or plasma protein binding of warfarin. Drugs or conditions increasing the anticoagulant effect of warfarin include: acetaminophen (tylenol), alcohol, allopurinol (zyloprim), paraaminosalicylic acid (PAS), anabolic steroids (adroyd, methandrostenolone, norethandrolone, stanolone), many antibiotics, antilipemic agents (clofibrate or atromid-S, d-thyroxine) aventyl, blood transfusion, cathartics, cincophen, diazoxide, disulfiram, drugs altering platelet function either by producing thrombocytopenia or altering platelet function, such as cytotoxic drugs, enzymes (bromelin and papain), ethacrynic acid, glucagon, hepatotoxins (carbon tetrachloride), mefenamic acid (ponstel), 6-mercaptopurine, methylthiouracil, monamine oxidase inhibitors, nalidixic acid, oxyphenobutazone (landearil), phenylbutazone (butazolidin), phenyramidol (analexin), prolonged hot weather, prolonged narcotic therapy, radioactive compounds, reserpine, salicylates (in excess of 1 gm/day), sulfinpyrazone, sulfonamides, sulfonylureas, x-ray therapy. Factors decreasing effect of oral anticoagulants include: diabetes mellitus, edema, hyperlipemia, lactation and many drugs — antacids, antihistamines, ascorbic acid, cholestyramine, corticosteroids, phenytoin, diuretics (mercurials and thiazides), ethychloroynol (placidyl), griseofulvin, haloperidol, mineral oil, oral contraceptives, barbiturates, and xanthines. This is an imposing list and reminds us of the importance of the many factors altering drug effects and of the need to carefully monitor coumarin therapy.

DR. OCHSNER: The hematuria occurred while the patient was on anticoagulants and was associated with discomfort in the form of left flank pain. There was no history of trauma. She is quite obese and trauma to the kidney would also be less likely to occur. The warfarin was discontinued and her urine grossly cleared despite the continuation of the left flank pain and discomfort. It was at this time that she was noted to have a large, left renal mass on an IVP. The lesion was distorting the collecting system and causing dilatation of the upper full calyces. The kidney mass was further confirmed by ultrasound and later by a CT scan. The CT scan strongly suggested a renal tumor; however, a hematoma or renal abscess was in the differential diagnosis. Beside the findings of the CT scan, there was other evidence suggesting a renal tumor, namely recurrent lower extremity thrombophlebitis, anemia and the low-grade fever without evidence of an infection. A

radical left nephrectomy was carried out. It was noted at operation that she had a large intrarenal hematoma without evidence of malignancy. The bleeding created a very interesting dissection line within the kidney. The cortex was separated from the renal pelvis and calyces throughout a large portion of the kidney, particularly the lower pole. Intrarenal hematoma secondary to anticoagulant has not been recognized before and a review of the literature has not revealed a similar case. This then is another form of hemorrhagic complication related to anticoagulation therapy.

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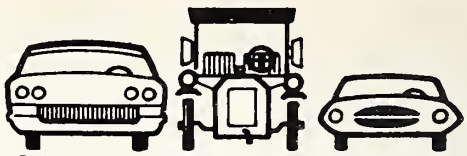

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JINGLE DOCS

Verse

Dashing Down the Halls
Of Sodak Hospitals
We Doctors Make Our Rounds
Day and Night All Year
We Smile, We Laugh, We Frown
With Patients Young and Old
Our Families Waiting Patiently
To Have Us Come Home

Chorus

Ents, Ob, Gyn, Surgeon, Gps Too
Ophtho, Ortho, X-Ray Men
Are In Our Family Too
Cardio, Gastro, Neuro, Uro, Peds and Path and Derm
Anesthesia, Internal and Pulmonary Too

At this time of the year, one has the privilege of poetic license — try this to the tune of **Jingle Bells!**

A MERRY CHRISTMAS AND A HAPPY NEW YEAR!

Howard L. Saylor, Jr., M.D., President
South Dakota State Medical Association

10th ANNUAL

BLACK HILLS WINTER SKI SEMINAR

ON COLORECTAL DISEASE AND FLEXIBLE SIGMOIDOSCOPY; REHABILITATION, ACUTE AND CHRONIC, CHILD AND ADULT; AND COMPUTERS IN OFFICE PRACTICE

JANUARY 31, FEBRUARY 1, 2, 1985

HOLIDAY INN OF THE NORTHERN BLACK HILLS, SPEARFISH, SOUTH DAKOTA

Hosted by the South Dakota Chapter of the American Academy of Family Physicians

This program has been reviewed and is acceptable for 12 prescribed hours by the American Academy of Family Physicians and 12 hours Category 1 AMA Physician Recognition Award.

WEDNESDAY, JANUARY 30, 1985

4:00 p.m. SDAFP Board of Directors Meeting

THURSDAY, JANUARY 31, 1985

MORNING SESSION

Charles Swanson, M.D., Moderator

7:00-7:30 a.m. Registration
Complimentary continental breakfast

7:30-8:10 a.m. *Proctosigmoidoscopy: Why? How? How Often? Techniques*
John D. Barker, Jr., M.D.

8:15-8:55 a.m. *Hemoccult: A Vital Screening Tool for Colorectal Cancer*
Robert Roberts, M.D.

9:00-9:40 a.m. *Colorectal Cancer*
John D. Barker, Jr., M.D.

9:45-5:00 p.m. WINTER SPORTS TIME

10:00-12:00 noon *Optional Lab on Flexible Sigmoidoscopy*
Morning speakers and Christine Bucy, M.D. and Verdayne Brandenburg, M.D.

3:00-5:00 p.m. *Optional Lab on Flexible Sigmoidoscopy*
Morning speakers and Christine Bucy, M.D. and Verdayne Brandenburg, M.D.

EVENING SESSION

Michael Brown, M.D., Moderator

5:00-5:30 p.m. Registration
Complimentary coffee, hot wine and hot buttered rum

5:30-6:10 p.m. *Pruritis Ani and Hemorrhoids*
Robert Roberts, M.D.

6:15-6:55 p.m. *Diverticular Disease*
John D. Barker, Jr., M.D.

7:00-7:45 p.m. *Irritable Bowel Syndrome*
Robert Roberts, M.D.

7:45 p.m. EVENING FREE

FRIDAY, FEBRUARY 1, 1985

MORNING SESSION

Richard Finley, M.D., Moderator

7:00-7:30 a.m. Registration
Complimentary continental breakfast

7:30-8:10 a.m. *Rehabilitation: Acute, Child*
Stephen F. Noll, M.D.

8:15-8:55 a.m. *Rehabilitation: Acute, Adult*
Frederick Entwistle, M.D.

9:00-9:45 a.m. *Rehabilitation: The Physical Therapy Role*
W. W. Blount, R.P.T.

9:45-5:00 p.m. WINTER SPORTS TIME

10:00-12:00 noon *Optional Real Estate Investment Seminar*
James D. Owen, C.P.M.

EVENING SESSION

Richard W. Honke, II, M.D., Moderator

5:00-5:30 p.m. Registration
Complimentary coffee, hot wine and hot buttered rum

5:30-6:10 p.m. *Rehabilitation: Chronic, Child*
Stephen F. Noll, M.D.

6:15-6:55 p.m. *Rehabilitation: Chronic, Adult*
Frederick Entwistle, M.D.

7:00-7:45 p.m. Panel, Friday Speakers

7:45 p.m. EVENING FREE

SATURDAY, FEBRUARY 2, 1985

MORNING SESSION

Richard McClaflin, M.D., Moderator

7:00-7:30 a.m. Registration
Complimentary continental breakfast

7:30-7:50 a.m. *A Basic Computer Literacy Session*
Judy Winter, M.A.

8:00-10:00 a.m. *A Seminar of Computer Vignettes*
Glen Aukerman, M.D.
Do I Need a Computer?
What Office Functions Should be Considered?
Which Systems Are Most Appropriate?
What Business Applications Make Sense for Family Physicians?
How to Use the Computer Clinically in Family Medicine.
Components and Basic Functions of the Computer System.
The Nature of Software: A Language for Man and Medicine.
National Data Bases and Medical Terminology with Coding.
The Next Five Years, What the Future Holds

10:00-12:00 noon *Optional Real Estate Investment Seminar*
James D. Owen, C.P.M.

12:00 noon SEMINAR CLOSES
WINTER SPORTS CONTINUE

MAKE PLANS TO ATTEND NOW, WRITE:
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SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105

RESIDENCY SIZE REDUCED

Earl D. Kemp, M.D., Program Director of the Sioux Falls Family Practice Residency, announced action by the FPC, Inc. Board of Directors which will reduce the size of South Dakota's only Family Practice Residency. This reduction from 12-12-12 to 8-8-8 will begin July 1, 1985.

MYTHICAL DECISION MAKING
An Editorial

Myth: There are too many doctors.

Facts: The Graduate Medical Education National Advisory Committee (GMENAC) report, released in 1980, concluded that by the 1990s there would be sufficient numbers of physicians in the United States for the nation's health care needs. This report also said that numbers of family physicians would be just adequate, but that geographic maldistribution would continue to be a problem in spite of the numbers. The 1979 Robert Wood Johnson report had said, "To solve geographic inequities will require some leeway in the supply to encourage health personnel to locate in areas that would not otherwise get the manpower required."

Continued federal funding of family practice supports the contention that family practice residency graduates are fulfilling the need by practicing in remote and rural areas.

Family practice and other "primary care" specialties are not synonymous.

Myth: There are too many family physicians in South Dakota.

Facts: Several carefully designed studies, applying numerical and geographic criteria, have continued to show the need for at least 20 family physicians per year to enter practice in South Dakota. Currently the ratio is one family physician per 4,200 population. Documented needs for at least one per 2,500 population have been made, however, with current health care trends (more ambulatory care, less hospital care, disease prevention/health promotion, prepaid programs) a more realistic number is one family physician per 1,500 to 2,000 population.

Nationally accepted criteria include one M.D. (all types of specialties) per 400-500 population. South Dakota currently has one per 750, Minnehaha County 1 per 400. However, even in Minnehaha County with a population of 115,000 and 40 family physicians, this ratio of one per 2,800 population is still below the documented need. And many of these are currently fulfilling an expanded role due to continued existing voids in several rural areas surrounding Minnehaha County.

National statistics for prepaid plans show that they can and will exist with one family physician per 1,000 enrollees, and it is projected that most medical care in prepaid plans will be rendered by family physicians.

A recent survey of family/general practitioners in South Dakota showed that two-thirds felt that there are not enough family physicians in South Dakota. They also felt that their opinions echoed the sentiment of their patients throughout the state.

Myth: There is no support for the program.

Facts: Every sponsoring hospital (2) medical staff vote has been supportive of the program and its size (12-12-12). This level of support has also been expressed by the South Dakota Academy of Family Physicians, the specialty society representing family practice in South Dakota.

A recent survey of family/general practitioners in South Dakota showed that over 95% of these physicians are supportive of the program and two-thirds felt that it should be continued at its present level.

The program has continued to receive ongoing legislative support since 1976.

Myth: Family practice will not survive as a needed specialty.

Facts: Family practice is the fastest growing medical specialty in the history of American medicine. Since its founding in 1969, more than 33,000 M.D.s hold current Diplomate certificates from the American Board of Family Practice, making this total number second only to Internal Medicine (founded in 1936). Its specialty society, the American Academy of Family Physicians, is the largest medical society in the world, with over 55,000 members.

It is reasonable to assume that 60% of all health care delivery in the **new environment** will be handled by family physicians — including providing the majority of geriatric care. One hundred million people will get their care from prepaid systems within the next ten years, and these family physicians (case managers) will also have to provide expertise as consultants for patients and families during complicated and often fragmented secondary and tertiary hospital care.

Myth: It is too costly.

Facts: Many community hospitals throughout the country have a long tradition in medical education. This program element has allowed them to provide better medical care through costs borne by their patients and the patients' "third party payers." Also, documented demographics show that many of their patients enter, by referral, from their earlier trainees, now practicing in the hospitals' "functional medical service areas."

Financial support that hospitals receive through federal programs (Medicare, Medicaid) draw on the taxpayer base of all citizens, both state and federal, transcending geopolitical boundaries.

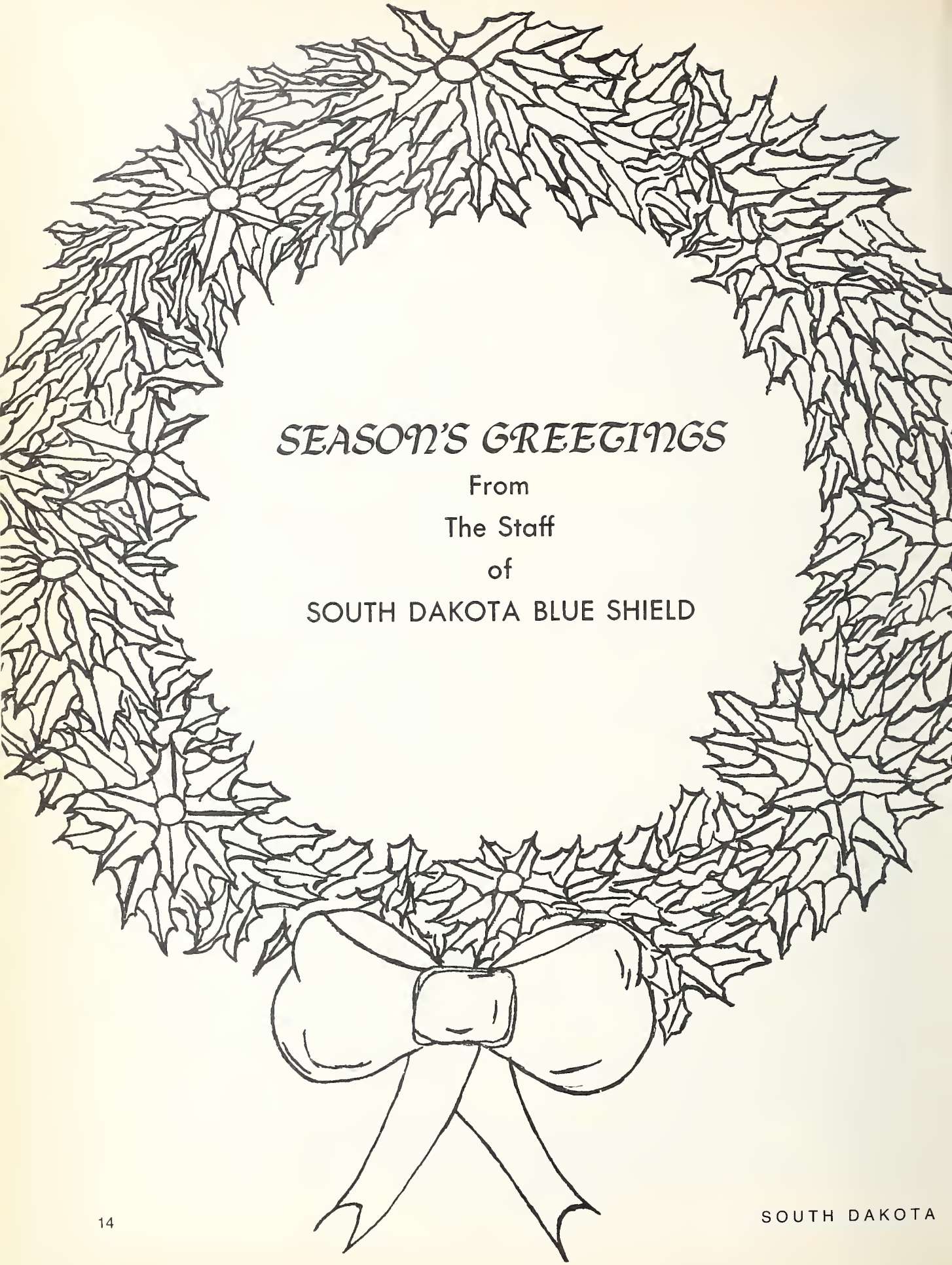
The new system of payments to hospitals for federally financed patient care (Medicare, Medicaid) allows continued, and even expanded, reimbursement for graduate medical education through direct and indirect educational allowances. While changes may occur, federal support of graduate medical education will continue in some fashion appropriate to supply graduate physicians for health care delivery in the United States.

What Does This Mean?

In spite of continuing and comprehensive studies showing both need and support, the size of the Sioux Falls Family Practice Residency Program has been reduced from 12-12-12 to 8-8-8, this regressive activity to begin July 1, 1985.

Mythical decision making.

Loren H. Amundson, M.D.
Editor, SDAFP Chapter News


SEASON'S GREETINGS
From
The Staff
of
SOUTH DAKOTA BLUE SHIELD

Adenocarcinoma of the Endometrium

Milton G. Mutch, Jr., M.D.*

ABSTRACT

The incidence of adenocarcinoma of the endometrium has been increasing over the past decade. Management of adenocarcinoma of the endometrium has changed over the past few years from preoperative irradiation followed by a simple hysterectomy to more current methods of individualization of therapy based on the clinical, surgical, and pathological findings. While the disease has a relatively good prognosis, even when caught early there is a group of early lesions that do poorly following a simple hysterectomy. This article attempts to outline that group of early adenocarcinomas that require individual and more specialized treatment.

Fifty-seven cases of adenocarcinoma, that were treated at Sioux Valley Hospital over the past five years, have been evaluated. The results and type of treatments were tabulated. Interestingly, the predominant number of cases have been stage I diseases. This indicates an awareness of the disease and early diagnosis.

A suggestion is also made regarding prophylaxis.

In the United States, cancer of the endometrium is the most common malignancy of the female pelvis. The American Cancer Society estimates approximately 38,000 women will develop carcinoma of the endometrium annually.[1] In reviewing the predicted incidence for the decade of the 1970's, the American Cancer Society noticed an increase of adenocarcinoma of the endometrium to 1.5 times the frequency as carcinoma of the cervix and of the ovary. The changing frequency has been suggested for several reasons, one of which is the increased incidence of unopposed exogenous estrogen.

Current survival figures for stage I endometrial cancer are reported as high as 70%.[2, 3, 4, 5, 6] Unfortunately, physicians have taken a somewhat complacent attitude towards adenocarcinoma of the endometrium. Rationale is that because of postmenopausal bleeding, diagnosis can be established at an early stage. Seventy-five percent of all adenocarcinomas in the endometrium are stage I. Because of this, there is a general feeling that the lesions can be cured by simple hysterectomy with reasonably good results. However, the 70% five year survival rate does not compare well with the stage I carcinoma of the cervix, which is approximately 90%. In recent years, an increasing concern has been directed toward that group of high risk patients in whom the possibility of salvage may have been jeopardized by the excellent treatment results observed in patients with early lesions and at low risk. The results of the Gynecologic Oncology groups investigation on the significance of the depth of uterine invasion, histological grade, and the geographical polarity within the uterus as determinance of the probability of nodal metastatic spread of this disease was studied. Creasman has found in stage IA lesions, a 6.2% metastasis to the pelvic nodes and 3.8% to the aortic nodes. IB lesions have 18% to the pelvic nodes and 11.7% to the aortic nodes. This also was noted in the evaluation of the grade of tumors. Grade I tumor showed a 3.1% to the pelvic nodes and 1.5% to the aortic nodes. Grade II tumors showed 10% to the pelvic nodes and 4% to the aortic nodes. Grade III tumors showed 36% to the pelvic nodes and 28% to the aortic nodes.

The study also shows that the invasion into the uterus increases the incidence of both pelvic and aortic node metastasis. This increase is reflected from 3.6% to the pelvic nodes when the lesion is confined to the endometrium to 43% when there is deep muscle invasion. When confined to the endometrium, 1.8% to the aortic nodes as compared to

* Obstetrician and Gynecologist, Sioux Falls. Clinical Professor in Obstetrics & Gynecology, USD School of Medicine, Sioux Falls, SD.

21% when deep muscle invasion is present.[7]

Creasman however, did note that aortic node spread was detected in only 2.7% of 36 cases with clinically negative nodes. Therefore, because of technical difficulties when aortic nodes are clinically negative, node sampling may not be justified in light of the apparent low incidence of metastasis.

The increased incidence of adenocarcinoma of the endometrium causes one to center on those patients who are at high-risk for endometrial cancer. The median age of cases of carcinoma of the endometrium is 61.1 years. It is found in women who are obese, nulliparous, late menopause, hypertension, diabetes, exposure to ionized radiation, estrogen producing tumors, polycystic ovarian disease and unopposed excess endogenous and exogenous estrogens.

Abnormal uterine bleeding is the cardinal symptom of adenocarcinoma of the endometrium, particularly in perimenopausal and postmenopausal patients. A high degree of suspicion for this disease must be entertained as 20-25% of corpus cancers are found in patients before menopause. Office diagnosis utilizes multiple cytologic and histologic techniques. The endometrial biopsy seems to be the most accurate study, having an accuracy rate of 62-90%.

Various brush and washing techniques have been used. However, the accuracy of these techniques is dependant on the ability of the pathologist to interpret the specimen and the ability of the clinician to obtain satisfactory tissue specimens.

Clinical staging prior to the initiation of therapy is performed using the International Federation's Gynecological Oncologists Classifications.[8] Tumor staging should include a fractional dilatation and curettage with a separate specimen from the endocervical canal and from the uterine corpus. Barium enema, I.V.P., chest x-ray, and other indicated pre-operative evaluations i.e., cystoscopy, proctoscopy, and ultrasound are ordered when indicated.

STAGING OF CARCINOMA OF THE CORPUS

Stage Characteristics

I Carcinoma confined to corpus.

IA Uterine cavity sounds less than 8cm.

IAG1—Highly differentiated adenomatous carcinomas

IAG2—Differentiated adenomatous carcinomas with partly solid areas

IAG3—Predominantly solid or entirely undifferentiated carcinomas

IB Uterine cavity sounds greater than 8cm.

IBG1—Highly differentiated adenomatous carcinomas

IBG2—Differentiated adenomatous carcinomas with partly solid areas

IBG3—Predominantly solid or entirely undifferentiated carcinomas

II Carcinoma has involved corpus and the cervix.

III Carcinoma has extended outside the uterus but not outside true pelvis.

IV Carcinoma has extended outside of true pelvis or has obviously involved the mucosa of the bladder or rectum. A bulbous edema as such does not permit allotment of a lesion to stage IV.

Recommended therapy for stage I lesions of adenocarcinoma of the endometrium is an abdominal hysterectomy with bilateral salpingo-oophorectomy and cellular washings of the pelvis and abdominal cavity with selective pelvic and aortic node biopsies in all cases.[9]

Adjuvant irradiation therapy of vaginal ovoids and external irradiation is then applied to those cases who have significant myometrial invasion, grade II-III tumors, or positive node biopsies from either pelvic or aortic nodes. Positive abdominal cytology exhibits an extremely poor prognosis and therapy consists of external irradiation or radio active phosphorous.[10]

Stage II disease however, is treated with preoperative irradiation, usually in the form of vaginal ovoids, followed by an abdominal hysterectomy and bilateral salpingo-oophorectomy and elective node biopsies. Adjuvant therapy includes external beam irradiation or hormonal therapy. Stage III lesions are treated with total abdominal hysterectomy, bilateral salpingo-oophorectomy, omentectomy, and postoperative irradiation and chemotherapy depending on the findings clinically at the time of surgery as well as the microscopic evaluations. In Stage IV lesions, when spread is limited to the pelvis, the patient should be treated with external x-ray treatments as well as intravaginal and intrauterine radium insertion before surgery.

REVIEW OF CASES AT SIOUX VALLEY HOSPITAL

Fifty-seven cases of adenocarcinoma of the endometrium were seen between January 1, 1968 and December 31, 1983. The files of our office and the pathology laboratory at Sioux Valley Hospital were examined to identify all cases. There was 100%

follow up with two patients succumbing to their disease, and seven are living with suspected disease and on progestational therapy.

Fifty-six patients received abdominal hysterectomy with bilateral salpingo-oophorectomy. One patient was seen for post-menopausal bleeding two years after she had been irradiated elsewhere for adenocarcinoma of the endometrium with no therapy because of a previous stroke and cardiac disease. A repeated dilatation and curettage on this patient showed no evidence of tumor.

Prior to 1980, 8 patients received pre-operative irradiation. Since 1980, surgical staging was instituted and 19 patients have received post-operative irradiation.

Hypogastric nodes were selectively biopsied in 28 patients with one positive node found. Aortic nodes were biopsied in 5 cases with 2 positive nodes found. Clinically, the nodes in all cases were examined. Cell washings were done in each case with 4 positive cytologies present. One patient with positive cytology received Cytoxan and Adriamycin regime with post-operative irradiation with a second-look and remains 49 months post-operation free of disease. Two patients with positive cytology have received post-operative chemotherapy and irradiation and are free of disease as of 15 and 18 months respectively. The fourth patient is 3 months post-surgery and on Cytoxan, Adriamycin, and 5FU therapy.

The distribution of the age of patients was:

AGE OF PATIENT	NUMBER OF PATIENTS
Less than 39	1
40-49	4
50-59	13
60-69	17
Greater than 70	21
	57

	STAGE				
	IA	IB	II	III	IV
Grade I	18	4	—	—	—
Grade II	13	12	1	—	1
Grade III	5	2	1	—	—
	36	18	2	0	1

Hospital stay is reflected in:

HOSPITAL STAY	NUMBER OF PATIENTS
Less than 11 days	43
12-17 days	11
18-28 days	3
	57

Survival rates:

MONTHS	NUMBER OF PATIENTS
Expired	2
Less than 12 months	20
12-24 months	12
25-48 months	10
Greater than 48 months	13
	57

CONCLUSIONS

Adenocarcinoma of the endometrium by virtue of its location, early warning signs of bleeding, and a relatively high cure rate of up to 70% has been treated with a cavalier fashion. Seventy-five percent of cases diagnosed are stage I. As we have shown, there are many variables in the staging and diagnosis that reflect the ultimate cure rate. The aim of successful treatment is improvement on those high risk groups by proper early diagnosis and adjuvant treatment.

In order to make a diagnosis, one must think of adenocarcinoma of the endometrium even in pre-menopausal women. Tissue must be obtained either by biopsy or differential dilatation and curettage. Proper staging must be done to assign proper therapy. Demonstration of nodal involvement more accurately defines the extent and the nature of such treatment. It is our belief that a profile of factors, including depth of invasion, tumor histological features, peritoneal cell washings, and stage of the disease provides a realistic framework determinable at the time of operation and by pathologic examination of the uterus on which to make the decision for further treatment.

Patients that are treated with estrogen hormonal therapy in the post-menopausal years would benefit by concomitant administration of a progestational agent. A five-fold reduction in the risk of adenocarcinoma of the endometrium is found if progestational agents are used in conjunction with estrogen therapy. A recommended regime is Premarin, .625mg, days 1-25 of each month followed by Aygestin, 5mg, days 15-25 of each month.

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"READY FOR CHRISTMAS"

"Ready for Christmas," she said with a sigh
As she gave a last touch to the gifts piled high,
Then wearily sat for a moment and read
Til soon, very soon, she was nodding her head.

Then quietly spoke a voice in her dream,
"Ready for Christmas? What do you mean?
Ready for Christmas, when only last week
You wouldn't acknowledge your friend on the street?

Ready for Christmas while holding a grudge,
Perhaps you had better let God be the judge.
Why, how can the Christ-Child come and abide
In a heart that is selfish and filled with pride.

Ready for Christmas, when only today
A beggar lad came and you turned him away
Without even a smile to show that you cared?
The little he asked — it could have been spared.

Ready for Christmas? You've worked, it's true.
But just doing the things that you wanted to do.
Ready for Christmas? Your circle's too small.
Why you are not ready for Christmas at all!"

She awoke with a start, and a cry of despair
There's so little time, and I've still to prepare!
O Father, forgive me, I see what you mean,
To be ready means more than a house swept clean!

Yes. More than the giving of gifts and a tree.
It's the heart swept clean that He wants to see.
A heart that is free from bitterness — sin.
Ready for Christmas, and READY FOR HIM!

Author unknown.

To those we love and see each day
And other loved ones far away
To all good friends whose friendship means so much
And those with whom we're somehow out of touch

MERRY CHRISTMAS EVERYONE!

Mary Wrage

Mary Wrage, President
South Dakota State Medical Association Auxiliary

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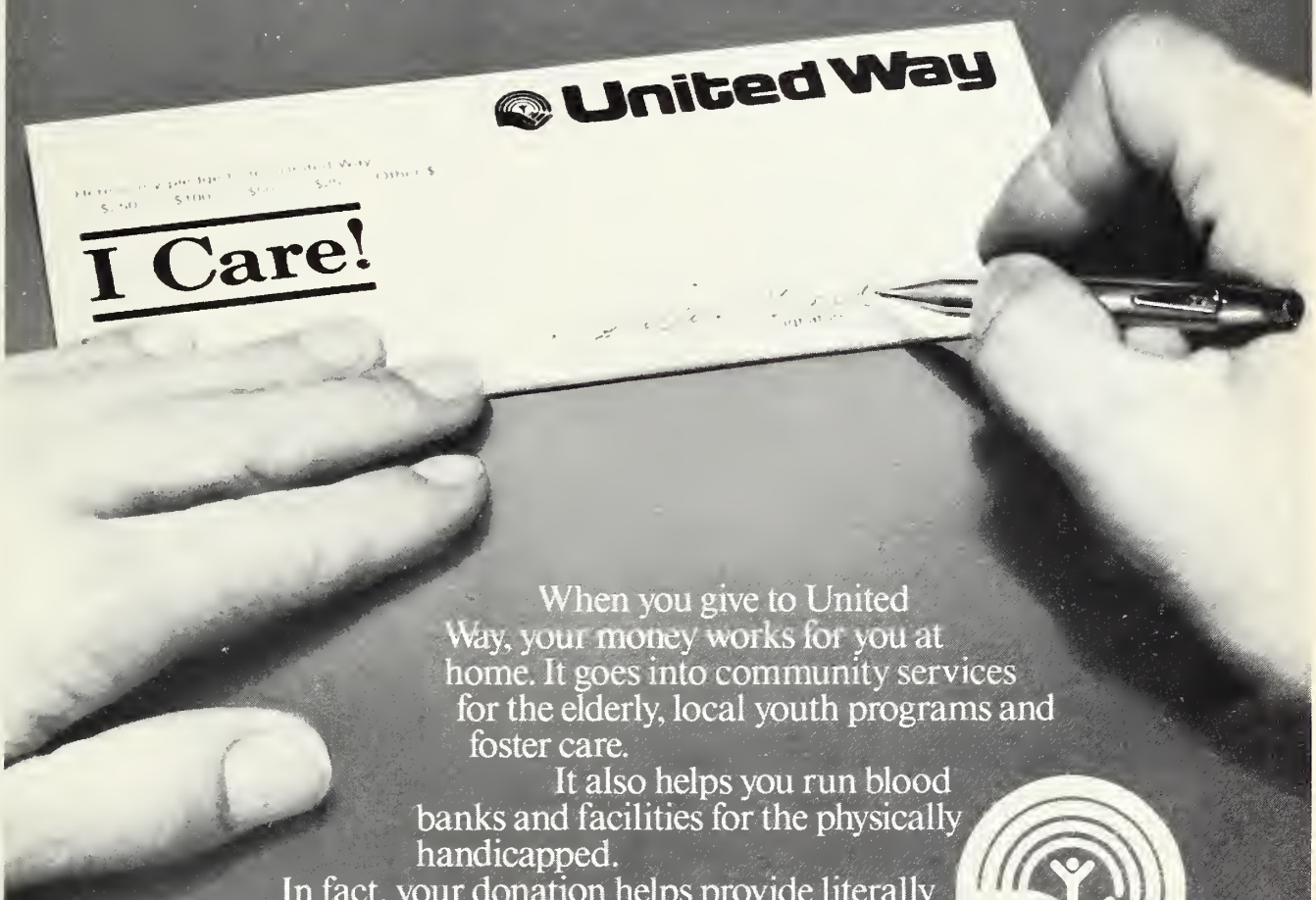

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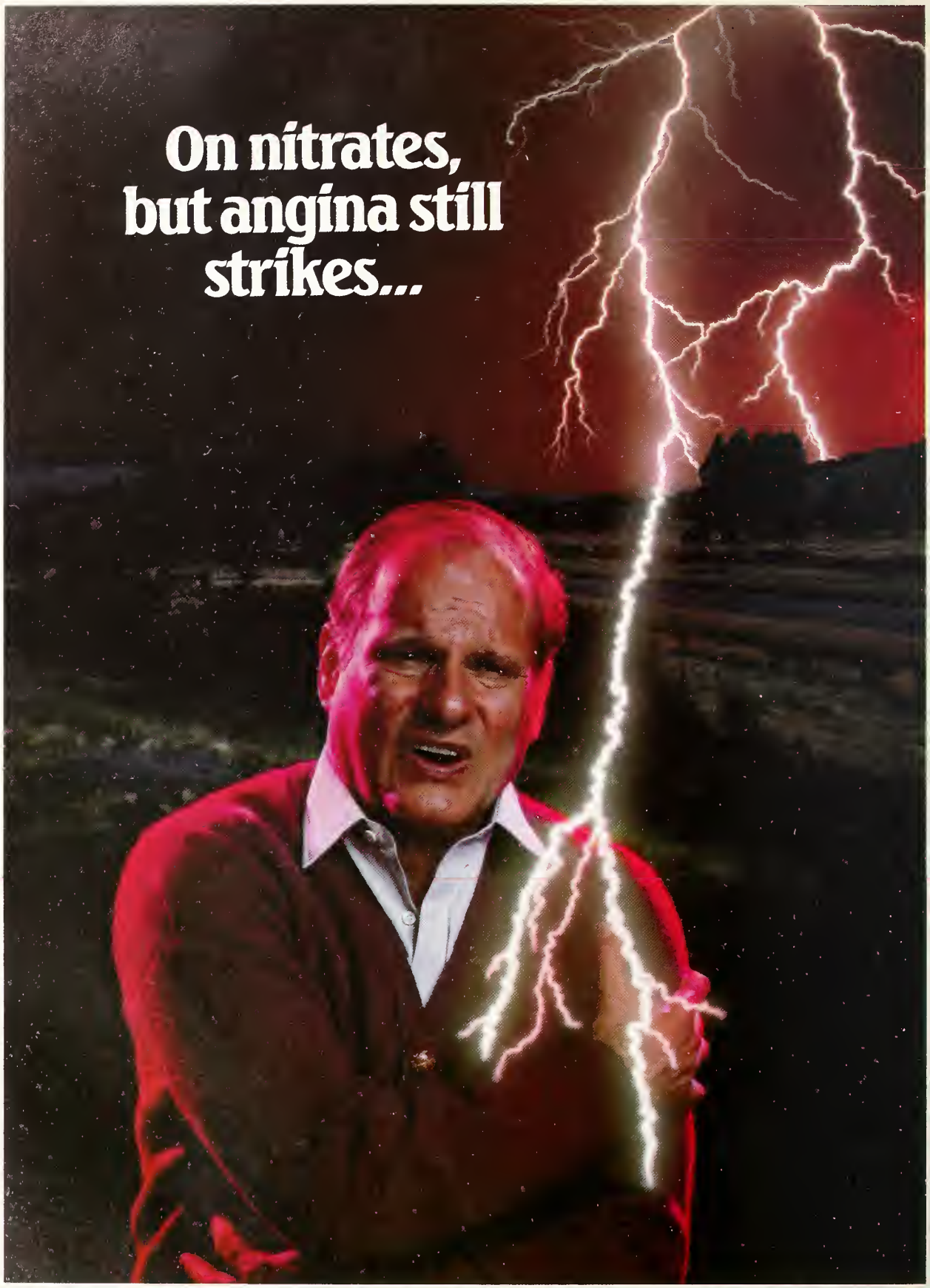
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Contraindications: Severe left ventricular dysfunction (see *Warnings*), hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction <30%) or moderate to severe symptoms of cardiac failure. Control milder heart failure with optimum digitalization and/or diuretics before ISOPTIN is used. ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild, and controlled by decrease in ISOPTIN dose). Occasional elevations of liver enzymes have been reported; patients receiving ISOPTIN should have liver enzymes monitored periodically. Patients with atrial flutter/fibrillation and an accessory AV pathway (e.g., W-P-W or L-G-L syndromes) may develop a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion. AV block may occur (3rd degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema, and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with ISOPTIN. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, patients should be monitored closely. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function or in patients who have also recently received methyldopa. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patient carefully monitored. ISOPTIN may have an additive hypotensive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C:* There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR<50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%). The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: confusion, paresthesia, insomnia, somnolence, equilibrium disorders, blurred vision, syncope, muscle cramps, shakiness, claudication, hair loss, maculae, and spotty menstruation. Overall continuation rate of 94.5% in 1,166 patients. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in 80 mg and 120 mg sugar-coated tablets. July 1982 2068

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Ventilation and Perfusion Lung Scans, Sioux Valley Hospital — 1981

W. A. Boade, M.D.*
M. C. Gullickson, CNMT†

ABSTRACT

Fifty-four pulmonary perfusion-only studies along with 237 combination ventilation and perfusion pulmonary studies were reviewed for the year 1981 at Sioux Valley Hospital, Sioux Falls, South Dakota. The conclusions indicate that perfusion-only lung scans in the age group of less than 35 years is probably adequate for evaluation of pulmonary thromboembolus. In the age groups greater than 35 years, however, the addition of the ventilation study is greatly helpful in interpreting the perfusion study, plus there is the addition of diagnostic entities with this procedure.

INTRODUCTION

In the recent past we have begun to look at nuclear isotope ventilation and perfusion scans done at Sioux Valley Hospital in Sioux Falls, South Dakota. We have questioned the need for both the ventilation and perfusion studies in all cases. We have also looked at the information obtainable from ventilation and perfusion studies. We retrospectively reviewed the ventilation and perfusion scans performed for a selected period of time. With this in mind we set about to evaluate the information obtained from all studies as listed below.

MATERIALS & METHODS

All pulmonary studies done in the calendar year 1981 were reviewed and categorized according to patient's age, scan results and whether the studies included perfusion scan only or ventilation and perfusion scan. Chest X-rays that were obtained and interpreted at the time of the isotope procedure were also reviewed. A total of 54 perfusion only isotope lung procedures and 237 ventilation perfusion isotope procedures were reviewed. Additional studies done as a part of a work-up prior to thoracic surgery for neoplasm (quantitative perfusion and ventilation studies) were not included in the present review.

Technetium — MAA (macroaggregated albumin) pulmonary scans were performed after an intravenous injection of a dose of 2.0 to 2.5 mCi 99M Tc MAA (Pyrolite by New England Nuclear) and 500 K count images utilizing anterior, posterior, right and left lateral views on a large field of view Anger gamma camera (Siemans Corp). Ventilation studies were performed by inhalation of approximately 10 mCi 133 Xenon dose administered through a (Pulmonex) Ventilation System. First breath and equilibrium breath images were obtained each requiring 20 second exposures followed by washout series of 20 second images for at least 3 minutes and longer if the tracer showed signs of trapping in the lungs on the camera oscilliscope. All 133 Xenon ventilation studies were recorded on a (ADAC) computer and the data was processed by creating six areas of interest (three over each lung) and time activity curves for each selected area. The curves were compared to comparable curves from the opposite side.

DATA ANALYSIS

In an overall abnormal to normal ratio, 32 of the 54 perfusion-only scans were abnormal for a 59% abnormality rate. Of the 237 cases in which ventilation and perfusion studies were performed, 165 were

* Nuclear Physician/Pathologist, Sioux Valley Hospital, Sioux Falls, SD.
† Nuclear Medicine Technologist, Sioux Valley Hospital, Sioux Falls, SD.

TABLE I
PERFUSION ONLY LUNG SCANS

Age	Under 35			35-55			Over 55		
P.E. Probability	Low	Mod	High	Low	Mod	High	Low	Mod	High
	0	0	0	1	0	0	6	10	6
COPD	Mild	Mod	Severe	Mild	Mod	Severe	Mild	Mod	Severe
	0	0	0	1	2	0	1	16	8
Pneumonia					1			8	

TABLE II
VENTILATION-PERFUSION LUNG SCANS

Age	35		35-55		55	
P.E. Probability	Low 0		0		2	
	Mod 0		3		4	
	High 1		3		21	
COPD	Vent Only	Vent/Perf	Vent Only	Vent/Perf	Vent Only	Vent/Perf
Mild	0	1	3	2	10	9
Mod	0	2	4	9	16	59
Severe	0	0	0	0	0	11
Pneumonia with COPD	0		3		17	
Pneumonia Only	4		1		10	

abnormal for a 70% abnormality rate. These abnormality rates reflect all disease states seen including pulmonary embolus, chronic lung disease COPD, neoplasm, atelectasis and pulmonary sequestration. There was no segregation of patient as to age in the overall abnormality rate.

In evaluating the scan results, the type of abnormality was considered and the severity of the abnormality rated. For pulmonary embolus the categorization was low, moderate or high probability. Another category was considered not suspicious for embolus. For COPD the categorization was mild, moderate or severe changes. Pneumonia was considered either present or absent. Studies were also subdivided into age categories of the following: under 35 years, 35-55 years, and over 55 years. In addition, studies which indicated only a single disease state were compared with studies demonstrating multiple disease states.

Pulmonary perfusion-only studies showed a ratio of 32 (59%) positive studies per 54 examinations performed (Table I). Of the 32 positive studies, 24 (75%) indicated pulmonary embolus (all categories). Six (25%) of these 24 indicated pulmonary embolus (all categories) only while 18 (75%) of the 24 indicated pulmonary embolus in addition to COPD and/or pneumonia. Of the remaining 8 positive perfusion-only studies, 6 (75%) indicated COPD plus pneumonia and 2 (25%) indicated pneumonia only.

When reviewing the studies by age category, only one (12%) of the eight patients studied in the under 35 year age group had an abnormal perfusion lung study. In the age 35 to 55 year group, two (40%) of five patients studied had an abnormal study. In the age 55 and older group 29 (75%) of 41 showed abnormal results.

In review of the 237 ventilation-perfusion studies, the addition of the ventilation study parameter yielded yet another category of abnormal interpretation. This was the normal perfusion with an abnormal ventilation study indicating COPD. The type of disease state was indicated along with severity of the disease state, and single or multiple disease states, and patient's age.

Of the 237 ventilation-perfusion studies performed 165 (70%) showed some form of abnormality. (Table II) Pulmonary embolus was indicated in 34 (20%) of the 165 abnormal studies. However, only 13 (30%) of the 34 positive for embolus studies suggested only pulmonary embolus. The remaining 21 were complicated by other disease processes such as chronic lung disease or pneumonia.

Chronic lung disease was evident in 126 (76%) of the 165 abnormal ventilation-perfusion scans. Of these, 33 (26%) cases were detected only in the ventilation portion of the study. Pneumonia was seen in 39 (16%) of the total ventilation-perfusion scans. Fifteen of the pneumonia cases showed no additional abnormalities to suggest other disease processes. Four patients studied showed evidence of neoplasm or pneumonia, one showed pulmonary sequestration, and one showed atelectasis with pneumonia. Four of the ventilation studies indicated emphysematous blebs.

Of academic interest only, 16 of the ventilation studies showed 133 Xenon activity in the liver indicating fatty metamorphosis.

TABLE III

Perfusion Lung Only				Ventilation/Perfusion Lung			
Age	35	35-55	55	Age	35	35-55	55
Normal	7	3	12	Normal	11	13	38
Abnormal	1	2	29	Abnormal	9	22	134
Total	8	5	41	Total	20	35	172
% Abnormal	12%	40%	75%	% Abnormal	45%	65%	78%

In reviewing ventilation perfusion studies on patient age basis, 9 (45%) out of 20 were abnormal in the under age 35 year group. (Table III) Twenty-two (63%) out of 35 studies were abnormal in the age group 35 to 55 years. In patients over 55 years, 134 (78%) out of 172 were abnormal.

CONCLUSIONS

Although the total number of studies in the perfusion-only category is small the trend seems to indicate that patients in a lower age bracket have less chance of additional abnormalities in their test interpretation than do the more elderly patients. This would appear to be verified by the ventilation-perfusion studies which show the abnormality rating increases sharply with age. To the imaging physician the addition of multiple parameters of pulmonary imaging aids in the interpretation of the study. It also contributes additional diagnostic abnormalities to the exclusion of pulmonary embolus, such as COPD, etc.

If a patient is less than 35 years of age, most likely we can assume that a perfusion lung scan and a chest x-ray is adequate for screening diagnosis for pulmonary embolus. If a patient is in the age group of 35-55, the possibility of additional complicating pulmonary disorders adds to the potential causes for perfusion abnormalities. With this in mind, ventilation studies are very helpful in excluding COPD, emphysematous blebs, neoplasm, etc. from the abnormalities which can cause perfusion deficits. This renders the pulmonary isotope scan (ventilation and perfusion) more specific with a higher rate of accuracy than with just a single perfusion lung scan, particularly in patients over the age of 35 years.

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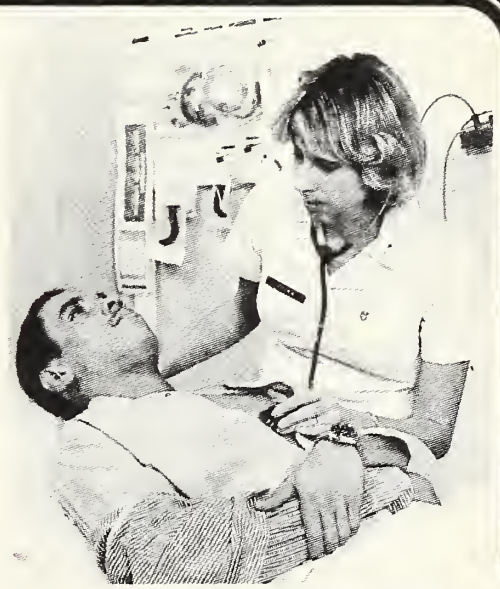

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Contraindications: Anaphylactoid reactions have occurred in individuals hypersensitive to *Motrin* Tablets or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin, iodides, or other nonsteroidal anti-inflammatory agents.

Warnings: Peptic ulceration and GI bleeding, sometimes severe, have been reported. Ulceration, perforation and bleeding may end fatally. An association has not been established. Use *Motrin* Tablets under close supervision in patients with a history of upper gastrointestinal tract disease, after consulting ADVERSE REACTIONS. In patients with active peptic ulcer and active rheumatoid arthritis, try nonulcerogenic drugs, such as gold. If *Motrin* Tablets are used, observe the patient closely for signs of ulcer perforation or GI bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity with papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with *Motrin* Tablets.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue *Motrin* Tablets and the patient should have an ophthalmologic examination, including central visual fields and color vision testing.

Fluid retention and edema have been associated with *Motrin* Tablets; use with caution in patients with a history of cardiac decompensation or hypertension. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of *Motrin* Tablets safety in patients with chronic renal failure have not been done.

Motrin Tablets can inhibit **platelet aggregation** and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and on anticoagulant therapy

Patients should report signs or symptoms of **gastrointestinal ulceration** or bleeding, skin rash, weight gain, or edema

Patients on prolonged **corticosteroid therapy** should have therapy tapered slowly when *Motrin* Tablets are added.

The antipyretic, anti-inflammatory activity of *Motrin* Tablets may mask inflammation and fever.

As with other nonsteroidal anti-inflammatory drugs, borderline elevations of liver tests may occur in up to 15% of patients. These abnormalities may progress, may remain essentially unchanged, or may be transient with continued therapy. Meaningful elevations of SGPT or SGOT (AST) occurred in controlled clinical trials in less than 1% of patients. Severe hepatic reactions, including jaundice and cases of fatal hepatitis, have been reported with ibuprofen as with other nonsteroidal anti-inflammatory drugs. If liver disease develops or if systemic manifestations occur (e.g. eosinophilia, rash, etc.), *Motrin* should be discontinued.

Drug interactions. *Aspirin:* used concomitantly may decrease *Motrin* blood levels.

Coumarin: bleeding has been reported in patients taking *Motrin* and coumarin.

Pregnancy and nursing mothers: *Motrin* should not be taken during pregnancy or by nursing mothers.

Adverse Reactions: The most frequent type of adverse reaction occurring with *Motrin* is gastrointestinal of which one or more occurred in 4% to 16% of the patients.

Incidence Greater than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence); **Central Nervous System:** Dizziness,* headache, nervousness; **Dermatologic:** Rash* (including maculopapular type), pruritus; **Special Senses:** Tinnitus; **Metabolic/Endocrine:** Decreased appetite; **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence less than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests; **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma; **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia, **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS); **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit; **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations; **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting; anaphylaxis; bronchospasm (see CONTRAINDICATIONS); **Renal:** Acute renal failure in patients with pre-existing significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria; **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence less than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis; **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri; **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions; **Special Senses:** Conjunctivitis, diplopia, optic neuritis; **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction; **Cardiovascular:** Arrhythmias (sinus tachycardia, sinus bradycardia); **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis; **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with *Motrin*. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under *"Probable Causal Relationship (PCR)"* if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under *"Causal Relationship Unknown"* if seven or more events have been reported but the criteria for PCR have not been met.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis. Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Do not exceed 2400 mg per day. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary.

Caution: Federal law prohibits dispensing without prescription.

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J-4791 August, 1984

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